Search history

Spivack 10/002526 06/15/2006

```
=>  d his full

     (FILE 'HOME' ENTERED AT 09:10:10 ON 15 JUN 2006)

     FILE 'HCAPLUS' ENTERED AT 09:10:22 ON 15 JUN 2006
                E US2005-002526/APPS
                E US2001-002526/APPS
                E US2001-02526/APPS
                E US2001-2526/APPS
L1            1 SEA ABB=ON  PLU=ON  US2001-2526/APPS
                D SCA
                E RADIATION SICKNESS+ALL/CT
L2         1818 SEA ABB=ON  PLU=ON  RADIATION SICKNESS/CT
                E RADIATION+ALL/CT
L3       462917 SEA ABB=ON  PLU=ON  RADIATION?/OBI
L4       781709 SEA ABB=ON  PLU=ON  ?RADIATION?/BI

     FILE 'REGISTRY' ENTERED AT 09:14:41 ON 15 JUN 2006
                E MESNA/CN
L5            1 SEA ABB=ON  PLU=ON  MESNA/CN
                D SCA

     FILE 'HCA' ENTERED AT 09:15:39 ON 15 JUN 2006

     FILE 'HCAPLUS' ENTERED AT 09:15:44 ON 15 JUN 2006
L6          546 SEA ABB=ON  PLU=ON  L5

     FILE 'STNGUIDE' ENTERED AT 09:16:24 ON 15 JUN 2006

     FILE 'REGISTRY' ENTERED AT 09:16:29 ON 15 JUN 2006
                E DIMESAN/CN
                E DIMESNA/CN
L7            1 SEA ABB=ON  PLU=ON  DIMESNA/CN
                D SCA

     FILE 'HCAPLUS' ENTERED AT 09:17:00 ON 15 JUN 2006
L8          104 SEA ABB=ON  PLU=ON  L7

     FILE 'STNGUIDE' ENTERED AT 09:17:08 ON 15 JUN 2006

     FILE 'HCAPLUS' ENTERED AT 09:17:27 ON 15 JUN 2006
L9          595 SEA ABB=ON  PLU=ON  L6 OR L8
L10          18 SEA ABB=ON  PLU=ON  L4 AND L9
                D SCA
L11       25113 SEA ABB=ON  PLU=ON  RADIOTHERAP?/BI
L12        9759 SEA ABB=ON  PLU=ON  RADIOPROTECT?/BI
L13          41 SEA ABB=ON  PLU=ON  (L11 OR L12) AND L9
L14          31 SEA ABB=ON  PLU=ON  L13 NOT L10
                D SCA

     FILE 'STNGUIDE' ENTERED AT 09:30:25 ON 15 JUN 2006

     FILE 'HCAPLUS' ENTERED AT 09:30:55 ON 15 JUN 2006
L15      989520 SEA ABB=ON  PLU=ON  ?RADIAT?/BI
L16          18 SEA ABB=ON  PLU=ON  L15 AND L9

     FILE 'STNGUIDE' ENTERED AT 09:31:31 ON 15 JUN 2006

     FILE 'HCAPLUS' ENTERED AT 09:46:32 ON 15 JUN 2006
                E THIOLS+ALL/CT
```

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L17      220762 SEA ABB=ON  PLU=ON  THIOLS+ALL/CT
                E DISULFIDES+ALL/CT
L18      272686 SEA ABB=ON  PLU=ON  DISULFIDES+ALL/CT
L19       15901 SEA ABB=ON  PLU=ON  (L17 OR L18) AND (L15 OR (L11 OR L12))

     FILE 'STNGUIDE' ENTERED AT 09:50:29 ON 15 JUN 2006

     FILE 'REGISTRY' ENTERED AT 10:00:30 ON 15 JUN 2006
L20             STRUCTURE UPLOADED
L21          28 SEA SSS SAM L20
                D STAT QUE L21
L22        5953 SEA SSS FUL L20
                DEL NWA377BATCH/A
                SAVE L22 SPI526STRG/A

     FILE 'HCAPLUS' ENTERED AT 10:09:34 ON 15 JUN 2006
L23        5378 SEA ABB=ON  PLU=ON  L22
L24         165 SEA ABB=ON  PLU=ON  (L15 OR (L11 OR L12)) AND L23

     FILE 'STNGUIDE' ENTERED AT 10:10:12 ON 15 JUN 2006

     FILE 'REGISTRY' ENTERED AT 10:10:37 ON 15 JUN 2006
L25        5952 SEA ABB=ON  PLU=ON  L22 NOT L5

     FILE 'HCAPLUS' ENTERED AT 10:10:55 ON 15 JUN 2006
L26        4986 SEA ABB=ON  PLU=ON  L25
L27         121 SEA ABB=ON  PLU=ON  (L15 OR (L11 OR L12)) AND L26

     FILE 'STNGUIDE' ENTERED AT 10:11:29 ON 15 JUN 2006

     FILE 'REGISTRY' ENTERED AT 10:17:00 ON 15 JUN 2006
L28             STRUCTURE UPLOADED
L29           6 SEA SUB=L22 SSS SAM L28
                D SCA
L30          69 SEA SUB=L22 SSS FUL L28
                SAVE L30 SPI526NOT1/A

     FILE 'STNGUIDE' ENTERED AT 10:20:59 ON 15 JUN 2006

     FILE 'REGISTRY' ENTERED AT 10:27:11 ON 15 JUN 2006
L31             STRUCTURE UPLOADED
L*** DEL        1 S L31 SAM SSS
L32             1 SEA SUB=L22 SSS SAM L31
                D SCA
L33             7 SEA SUB=L22 SSS FUL L31
                SAVE TEMP L33 SPI526NOT2/A
                SAVE  L33 SPI526NOT2/A
                D SCA L33

     FILE 'STNGUIDE' ENTERED AT 10:31:03 ON 15 JUN 2006

     FILE 'REGISTRY' ENTERED AT 10:34:51 ON 15 JUN 2006
L34             STRUCTURE UPLOADED
L35           1 SEA SUB=L22 SSS SAM L34
                D SCA
L36          34 SEA SUB=L22 SSS FUL L34
                SAVE L36 SPI526NOT3/A
                D SCA
                D COST
L37        5845 SEA ABB=ON  PLU=ON  L22 NOT (L5 OR L30 OR L33 OR L36)
```

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     FILE 'HCAPLUS' ENTERED AT 10:41:30 ON 15 JUN 2006
L38         3930 SEA ABB=ON  PLU=ON  L37
L39           68 SEA ABB=ON  PLU=ON  ((L11 OR L12) OR L15) AND L38
L40              ANALYZE PLU=ON  L39 1- RN :    2125 TERMS
                 D

     FILE 'REGISTRY' ENTERED AT 10:46:18 ON 15 JUN 2006
L41             1 SEA ABB=ON  PLU=ON  88859-04-5
                 D SCA

     FILE 'HCAPLUS' ENTERED AT 10:47:53 ON 15 JUN 2006
                 SEL RN L39
                 DELETE SELECT
                 SEL RN L39 1-14

     FILE 'REGISTRY' ENTERED AT 10:48:41 ON 15 JUN 2006
L42           968 SEA ABB=ON  PLU=ON  (2809-21-4/BI OR 40391-99-9/BI OR 66376-36-
                  1/BI OR 88859-04-5/BI OR 89987-06-4/BI OR 10596-23-3/BI OR
                  112809-51-5/BI OR 114084-78-5/BI OR 120511-73-1/BI OR 123948-87
                  -8/BI OR 1306-23-6/BI OR 13598-36-2/BI OR 16208-51-8/BI OR
                  23214-92-8/BI OR 305-03-3/BI OR 33069-62-4/BI OR 57-22-7/BI OR
                  59-02-9/BI OR 59-05-2/BI OR 10540-29-1/BI OR 105462-24-6/BI OR
                  107868-30-4/BI OR 114977-28-5/BI OR 118072-93-8/BI OR 119-13-1/
                  BI OR 13010-47-4/BI OR 131384-38-8/BI OR 143011-72-7/BI OR
                  147-94-4/BI OR 148-03-8/BI OR 148-82-3/BI OR 152459-95-5/BI OR
                  154-42-7/BI OR 154361-50-9/BI OR 15663-27-1/BI OR 162011-90-7/B
                  I OR 184475-35-2/BI OR 19767-45-4/BI OR 25322-68-3/BI OR
                  302-79-4/BI OR 33419-42-0/BI OR 3778-73-2/BI OR 4291-63-8/BI
                  OR 4342-03-4/BI OR 50-02-2/BI OR 52-24-4/BI OR 5300-03-8/BI OR
                  54083-22-6/BI OR 60-23-1/BI OR 63612-50-0/BI OR 645-05-6/BI OR
                  671-16-9/BI OR 70-18-8/BI OR 7440-21-3/BI OR 7616-22-0/BI OR
                  81627-83-0/BI OR 83-43-2/BI OR 85622-93-1/BI OR 89778-26-7/BI
                  OR 9000-81-1/BI OR 9003-01-4/BI OR 90357-06-5/BI OR 95058-81-4/
                  BI OR 97682-44-5/BI OR 10087-89-5/BI OR 101526-83-4/BI OR
                  10212-20-1/BI OR 10238-21-8/BI OR 103-90-2/BI OR 103628-46-2/BI
                   OR 104227-87-4/BI OR 106-45-6/BI OR 106603-90-1/BI OR
                  108560-70-9/BI OR 11000-17-2/BI OR 11003-32-0/BI OR 11003-33-1/
                  BI OR 110042-95-0/BI OR 110230-98-3/BI OR 11027-63-7/BI OR
                  110417-88-4/BI OR 11076-50-9/BI OR 111-20-6/BI OR 111-30-8/BI
                  OR 111-90-0/BI OR 111358-88-4/BI OR 11138-42-4/BI OR 112-61-8/B
                  I OR 112455-84-2/BI OR 112522-64-2/BI OR 112887-68-0/BI OR
                  113-15-5/BI OR 114-07-8/BI OR 114560-48-4/BI OR 114899-77-3/BI
                  OR 115256-11-6/BI OR 1156-19-0/BI OR 115956-12-2/BI OR
                  117048-59-6/BI OR 119804-96-5/BI OR 12063-98-8/BI OR 12064-03-8
                  /BI OR 12068-90-5/BI OR 120685-11-2/BI OR 121368-58-9/BI OR
                  121679-13-8/BI OR 122110-53-6/BI OR
L43             7 SEA ABB=ON  PLU=ON  L37 AND L42
                 D SCA
L44          5838 SEA ABB=ON  PLU=ON  L37 NOT L43

     FILE 'HCAPLUS' ENTERED AT 10:52:31 ON 15 JUN 2006
L45         3063 SEA ABB=ON  PLU=ON  L44
L46           34 SEA ABB=ON  PLU=ON  ((L11 OR L12) OR L15) AND L45

     FILE 'REGISTRY' ENTERED AT 10:55:46 ON 15 JUN 2006
                 E ETHANESULFONIC ACID, 2-MERCAPTO- /CN
L47             1 SEA ABB=ON  PLU=ON  ETHANESULFONIC ACID, 2-MERCAPTO- /CN
                 D SCA
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     FILE 'REGISTRY' ENTERED AT 10:56:30 ON 15 JUN 2006
                D IDE L5
                D IDE L47
                D IDE L7

     FILE 'STNGUIDE' ENTERED AT 10:59:59 ON 15 JUN 2006

     FILE 'REGISTRY' ENTERED AT 11:01:21 ON 15 JUN 2006

     FILE 'HCAPLUS' ENTERED AT 11:01:30 ON 15 JUN 2006
L48           359 SEA ABB=ON  PLU=ON  L47
L49             8 SEA ABB=ON  PLU=ON  L48 AND L39

     FILE 'REGISTRY' ENTERED AT 11:04:20 ON 15 JUN 2006
L50          5844 SEA ABB=ON  PLU=ON  L22 NOT (L5 OR L30 OR L33 OR L36 OR L47)

     FILE 'HCAPLUS' ENTERED AT 11:04:56 ON 15 JUN 2006
L51          3662 SEA ABB=ON  PLU=ON  L50
L52            60 SEA ABB=ON  PLU=ON  L51 AND ((L11 OR L12) OR L15)

     FILE 'MEDLINE' ENTERED AT 11:06:13 ON 15 JUN 2006

     FILE 'REGISTRY' ENTERED AT 11:06:38 ON 15 JUN 2006
L53            13 SEA ABB=ON  PLU=ON  L50 AND MEDLINE/LC
                  D SCA

     FILE 'MEDLINE' ENTERED AT 11:09:43 ON 15 JUN 2006
L54           462 SEA ABB=ON  PLU=ON  L53

     FILE 'STNGUIDE' ENTERED AT 11:10:09 ON 15 JUN 2006

     FILE 'MEDLINE' ENTERED AT 11:12:35 ON 15 JUN 2006
L55         51078 SEA ABB=ON  PLU=ON  RADIATION-PROTECTIVE AGENTS+NT/CT
L56        368763 SEA ABB=ON  PLU=ON  ?RADIAT?
L57            10 SEA ABB=ON  PLU=ON  L54 AND L55
L58            35 SEA ABB=ON  PLU=ON  L54 AND L56
L59            34 SEA ABB=ON  PLU=ON  L58 NOT L57
                  D TRIAL 1-5
                  D TRIAL 6-15
                  D TRIAL L57 1-10
                  E RADIATION, IONIZING+ALL/CT
L60         32292 SEA ABB=ON  PLU=ON  RADIATION, IONIZING+NT/CT
L61             1 SEA ABB=ON  PLU=ON  L60 AND L54
                  D TRIAL

     FILE 'REGISTRY' ENTERED AT 11:19:59 ON 15 JUN 2006
L62             1 SEA ABB=ON  PLU=ON  MAFOSFAMIDE/CN
                  D SCA
L63            12 SEA ABB=ON  PLU=ON  L53 NOT L62

     FILE 'MEDLINE' ENTERED AT 11:21:11 ON 15 JUN 2006
L64           244 SEA ABB=ON  PLU=ON  L63
L65             4 SEA ABB=ON  PLU=ON  L64 AND L55
L66             9 SEA ABB=ON  PLU=ON  L64 AND L56
L67             0 SEA ABB=ON  PLU=ON  L64 AND L60
L68            12 SEA ABB=ON  PLU=ON  (L65 OR L66)
                  D TRIAL 1-12

     FILE 'REGISTRY' ENTERED AT 11:24:19 ON 15 JUN 2006
                SEL NAME L50
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     FILE 'REGISTRY' ENTERED AT 11:26:27 ON 15 JUN 2006
L69             7 SEA ABB=ON  PLU=ON  L50 AND EMBASE/LC
                  D SCA
L*** DEL        0 S L69 AND NRS<1
L*** DEL        0 S L50 AND NR<1
L*** DEL        0 S L50 AND RC<1
L*** DEL        0 S L69 AND NR=0
L70             4 SEA ABB=ON  PLU=ON  L69 AND RSD/FA
                  D SCA
L71             3 SEA ABB=ON  PLU=ON  L69 NOT L70
                  D SCA

     FILE 'EMBASE' ENTERED AT 11:32:20 ON 15 JUN 2006
L72            21 SEA ABB=ON  PLU=ON  L71
L73        294525 SEA ABB=ON  PLU=ON  ?RADIAT?
                  E RADIATION PROTECTIVE AGENT+ALL/CT
                  E E2+ALL
L74          6762 SEA ABB=ON  PLU=ON  RADIOPROTECTIVE AGENT+ALL/CT
L75             5 SEA ABB=ON  PLU=ON  L72 AND (L73 OR L74)
                  D TRIAL 1-5

     FILE 'REGISTRY' ENTERED AT 11:35:33 ON 15 JUN 2006
                  SET SMARTSELECT ON
L76               SEL PLU=ON  L71 1- CHEM :      17 TERMS
                  SET SMARTSELECT OFF

     FILE 'EMBASE' ENTERED AT 11:35:33 ON 15 JUN 2006
L77           352 SEA ABB=ON  PLU=ON  L76
L78            17 SEA ABB=ON  PLU=ON  L77 AND L74
                  D TRIAL 1-17

     FILE 'MEDLINE' ENTERED AT 11:37:28 ON 15 JUN 2006

     FILE 'REGISTRY' ENTERED AT 11:37:36 ON 15 JUN 2006
                  D SCA L53
L79             1 SEA ABB=ON  PLU=ON  L53 AND S=6
L80             1 SEA ABB=ON  PLU=ON  L53 AND C3H8O3S2/MF
L81             2 SEA ABB=ON  PLU=ON  (L79 OR L80)

     FILE 'MEDLINE' ENTERED AT 11:40:24 ON 15 JUN 2006

     FILE 'REGISTRY' ENTERED AT 11:40:29 ON 15 JUN 2006
                  SET SMARTSELECT ON
L82               SEL PLU=ON  L81 1- CHEM :       4 TERMS
                  SET SMARTSELECT OFF

     FILE 'MEDLINE' ENTERED AT 11:40:29 ON 15 JUN 2006
L83           219 SEA ABB=ON  PLU=ON  L82
L84             6 SEA ABB=ON  PLU=ON  (L55 OR L56 OR L60) AND L83
                  D TRIAL 1-6
                  D COST

     FILE 'REGISTRY' ENTERED AT 11:43:39 ON 15 JUN 2006
L85            14 SEA ABB=ON  PLU=ON  L50 AND BIOSIS/LC
                  D SCA
L86             8 SEA ABB=ON  PLU=ON  L85 AND RSD/FA
L87             6 SEA ABB=ON  PLU=ON  L85 NOT L86

     FILE 'BIOSIS' ENTERED AT 11:46:09 ON 15 JUN 2006
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     FILE 'REGISTRY' ENTERED AT 11:46:16 ON 15 JUN 2006
                SET SMARTSELECT ON
L88             SEL PLU=ON  L87 1- CHEM :      25 TERMS
                SET SMARTSELECT OFF

     FILE 'BIOSIS' ENTERED AT 11:46:18 ON 15 JUN 2006
L89          523 SEA ABB=ON  PLU=ON  L88
L90       462658 SEA ABB=ON  PLU=ON  ?RADIAT?
L91            2 SEA ABB=ON  PLU=ON  L89 AND L90
L92        70394 SEA ABB=ON  PLU=ON  ?RADIOTHERAP?
L93         8062 SEA ABB=ON  PLU=ON  ?RADIOPROTECT?
L94            0 SEA ABB=ON  PLU=ON  L89 AND (L92 OR L93)

     FILE 'STNGUIDE' ENTERED AT 11:47:29 ON 15 JUN 2006

     FILE 'HCAPLUS' ENTERED AT 11:47:39 ON 15 JUN 2006
                D STAT QUE L52

     FILE 'MEDLINE' ENTERED AT 11:47:55 ON 15 JUN 2006
                D QUE NOS L65
                D QUE NOS L66
                D QUE NOS L84
L95            17 SEA ABB=ON  PLU=ON  L65 OR L66 OR L84

     FILE 'EMBASE' ENTERED AT 11:48:51 ON 15 JUN 2006
                D QUE NOS L78

     FILE 'BIOSIS' ENTERED AT 11:49:07 ON 15 JUN 2006
                D QUE NOS L91
                D QUE NOS L94
L96             2 SEA ABB=ON  PLU=ON  L91 OR L94

     FILE 'HCAPLUS, MEDLINE, EMBASE, BIOSIS' ENTERED AT 11:49:48 ON 15 JUN 2006
L97            90 DUP REM L52 L95 L78 L96 (6 DUPLICATES REMOVED)
                      ANSWERS '1-60' FROM FILE HCAPLUS
                      ANSWERS '61-74' FROM FILE MEDLINE
                      ANSWERS '75-89' FROM FILE EMBASE
                      ANSWER '90' FROM FILE BIOSIS
                   D IBIB ABS HITIND HITSTR L97 1-60
                   D IALL L97 61-90


     FILE HOME

     FILE HCAPLUS

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     FILE COVERS 1907 - 15 Jun 2006  VOL 144 ISS 25
     FILE LAST UPDATED: 14 Jun 2006  (20060614/ED)
```

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FILE REGISTRY
Property values tagged with IC are from the ZIC/VINITI data file provided by InfoChem.

STRUCTURE FILE UPDATES: 14 JUN 2006 HIGHEST RN 887828-19-5
DICTIONARY FILE UPDATES: 14 JUN 2006 HIGHEST RN 887828-19-5

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH January 6, 2006

Please note that search-term pricing does apply when conducting SmartSELECT searches.

****
* *
* The CA roles and document type information have been removed from *
* the IDE default display format and the ED field has been added, *
* effective March 20, 2005. A new display format, IDERL, is now *
* available and contains the CA role and document type information. *
* *
****

Structure search iteration limits have been increased. See HELP SLIMITS for details.

REGISTRY includes numerically searchable data for experimental and predicted properties as well as tags indicating availability of experimental property data in the original document. For information on property searching in REGISTRY, refer to:

http://www.cas.org/ONLINE/UG/regprops.html

FILE HCA

FILE COVERS 1907 - 8 Jun 2006 VOL 144 ISS 25
FILE LAST UPDATED: 8 Jun 2006 (20060608/ED)

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This file contains CAS Registry Numbers for easy and accurate substance identification.

FILE STNGUIDE

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Jun 9, 2006 (20060609/UP).

FILE MEDLINE
FILE LAST UPDATED: 14 JUN 2006 (20060614/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 is now (26 Feb.) available. For details on the 2006 reload, enter HELP RLOAD at an arrow prompt (=>).
See also:

http://www.nlm.nih.gov/mesh/
http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html
http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2006 vocabulary.

This file contains CAS Registry Numbers for easy and accurate substance identification.

FILE EMBASE
FILE COVERS 1974 TO 15 Jun 2006 (20060615/ED)

EMBASE has been reloaded. Enter HELP RLOAD for details.

EMBASE is now updated daily. SDI frequency remains weekly (default) and biweekly.

This file contains CAS Registry Numbers for easy and accurate substance identification.

FILE BIOSIS
FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 14 June 2006 (20060614/ED)

=>

=> file registry
FILE 'REGISTRY' ENTERED AT 10:56:30 ON 15 JUN 2006

Property values tagged with IC are from the ZIC/VINITI data file
provided by InfoChem.

STRUCTURE FILE UPDATES: 14 JUN 2006 HIGHEST RN 887828-19-5
DICTIONARY FILE UPDATES: 14 JUN 2006 HIGHEST RN 887828-19-5

New CAS Information Use Policies, enter HELP USAGETERMS for details.

TSCA INFORMATION NOW CURRENT THROUGH January 6, 2006

Please note that search-term pricing does apply when
conducting SmartSELECT searches.

```
**********************************************************************
*                                                                    *
* The CA roles and document type information have been removed from *
* the IDE default display format and the ED field has been added,   *
* effective March 20, 2005.  A new display format, IDERL, is now    *
* available and contains the CA role and document type information. *
*                                                                    *
**********************************************************************
```

Structure search iteration limits have been increased. See HELP SLIMITS
for details.

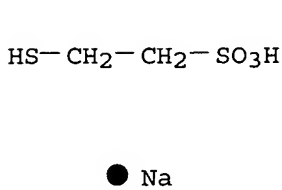
REGISTRY includes numerically searchable data for experimental and
predicted properties as well as tags indicating availability of
experimental property data in the original document. For information
on property searching in REGISTRY, refer to:

http://www.cas.org/ONLINE/UG/regprops.html

=> d ide L5

L5 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2006 ACS on STN
RN 19767-45-4 REGISTRY
ED Entered STN: 16 Nov 1984
CN Ethanesulfonic acid, 2-mercapto-, monosodium salt (8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN 2-Mercapto-1-ethanesulfonic acid monosodium salt
CN 2-Mercaptoethanesulfonic acid monosodium salt
CN 2-Mercaptoethanesulfonic acid sodium salt
CN D 7093
CN Mesna
CN Mesnex
CN Mesnum
CN Mistabron
CN Mistabronco
CN Mitexan
CN Mucofluid
CN Prehepon
CN Sodium 2-mercaptoethanesulfonate
CN UCB 3983

CN Uromitexan
DR 122504-78-3
MF C2 H6 O3 S2 . Na
CI COM
LC STN Files: ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, BEILSTEIN*, BIOSIS, BIOTECHNO, CA, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, DDFU, DRUGU, EMBASE, IFICDB, IFIPAT, IFIUDB, IMSCOSEARCH, IPA, MEDLINE, MRCK*, MSDS-OHS, PIRA, PROMT, PS, RTECS*, SYNTHLINE, TOXCENTER, USAN, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)
CRN (3375-50-6)



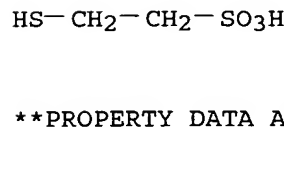
● Na

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

546 REFERENCES IN FILE CA (1907 TO DATE)
9 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
546 REFERENCES IN FILE CAPLUS (1907 TO DATE)

=> d ide L47

L47 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2006 ACS on STN
RN 3375-50-6 REGISTRY
ED Entered STN: 16 Nov 1984
CN **Ethanesulfonic acid, 2-mercapto- (6CI, 7CI, 8CI, 9CI)** (CA INDEX NAME)
OTHER NAMES:
CN β-Mercaptoethanesulfonic acid
CN 2-Mercaptoethanesulfonic acid
CN 2-Mercaptoethylsulfonic acid
CN Mercaptoethanesulfonic acid
CN Reduced coenzyme M
FS 3D CONCORD
MF C2 H6 O3 S2
CI COM
LC STN Files: ADISINSIGHT, AGRICOLA, ANABSTR, BEILSTEIN*, BIOSIS, BIOTECHNO, CA, CAOLD, CAPLUS, CASREACT, CHEMCATS, CHEMINFORMRX, CHEMLIST, CSCHEM, EMBASE, GMELIN*, MEDLINE, PS, SCISEARCH, TOXCENTER, USAN, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)

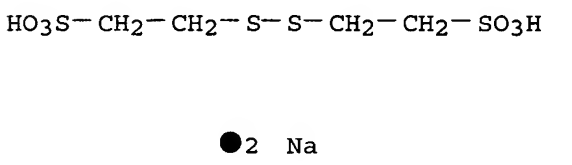


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

359 REFERENCES IN FILE CA (1907 TO DATE)
37 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
359 REFERENCES IN FILE CAPLUS (1907 TO DATE)
6 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=> d ide L7

L7 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2006 ACS on STN
RN 16208-51-8 REGISTRY
ED Entered STN: 16 Nov 1984
CN Ethanesulfonic acid, 2,2'-dithiobis-, disodium salt (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Ethanesulfonic acid, 2,2'-dithiodi-, disodium salt (6CI, 8CI)
OTHER NAMES:
CN 2,2'-Dithiodi-1-ethanesulfonic acid disodium salt
CN Bis(2-sulfoethyl)disulfide disodium salt
CN BNP 7787
CN **Dimesna**
CN Disodium 2,2'-dithiobis(ethanesulfonate)
CN Disodium 2,2'-dithiodiethanesulfonate
CN NSC 716976
MF C4 H10 O6 S4 . 2 Na
LC STN Files: ADISINSIGHT, ADISNEWS, ANABSTR, BIOSIS, CA, CAOLD, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMLIST, CIN, DDFU, DRUGU, EMBASE, IFICDB, IFIPAT, IFIUDB, IMSDRUGNEWS, IMSPATENTS, IMSRESEARCH, IPA, PHAR, PROMT, PROUSDDR, SYNTHLINE, TOXCENTER, USAN, USPAT2, USPATFULL
Other Sources: EINECS**, NDSL**, TSCA**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)
CRN (45127-11-5)



●2 Na

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

104 REFERENCES IN FILE CA (1907 TO DATE)
104 REFERENCES IN FILE CAPLUS (1907 TO DATE)
1 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

=> => file hcaplus
FILE 'HCAPLUS' ENTERED AT 11:47:39 ON 15 JUN 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.

FILE COVERS 1907 - 15 Jun 2006 VOL 144 ISS 25
FILE LAST UPDATED: 14 Jun 2006 (20060614/ED)

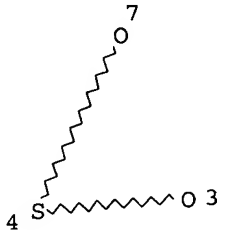
New CAS Information Use Policies, enter HELP USAGETERMS for details.

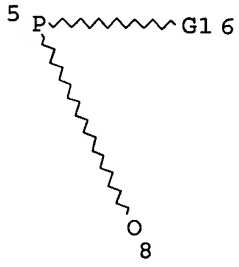
This file contains CAS Registry Numbers for easy and accurate substance identification.
'OBI' IS DEFAULT SEARCH FIELD FOR 'HCAPLUS' FILE

=> d stat que L52

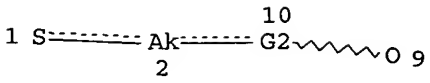
L5	1	SEA FILE=REGISTRY ABB=ON PLU=ON	MESNA/CN
L11	25113	SEA FILE=HCAPLUS ABB=ON PLU=ON	RADIOTHERAP?/BI
L12	9759	SEA FILE=HCAPLUS ABB=ON PLU=ON	RADIOPROTECT?/BI
L15	989520	SEA FILE=HCAPLUS ABB=ON PLU=ON	?RADIAT?/BI
L20		STR	

O 11 S 12





Page 1-A



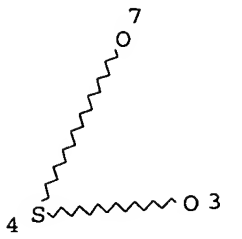
Page 2-A
VAR G1=11/12
VAR G2=4-2 4-9/5-2 5-9
NODE ATTRIBUTES:
NSPEC IS C AT 1
NSPEC IS C AT 2
NSPEC IS C AT 3

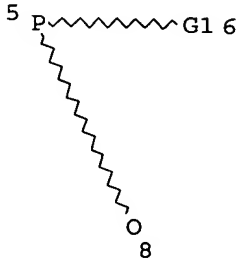
```
NSPEC   IS C      AT   4
NSPEC   IS C      AT   5
NSPEC   IS C      AT   6
NSPEC   IS C      AT   7
NSPEC   IS C      AT   8
NSPEC   IS C      AT   9
NSPEC   IS C      AT  10
CONNECT IS X2  RC AT   1
CONNECT IS X3  RC AT   2
CONNECT IS E1  RC AT   9
DEFAULT MLEVEL IS ATOM
MLEVEL  IS CLASS  AT   1  2  3  4  5  7  8  9 11 12
DEFAULT ECLEVEL IS LIMITED

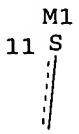
GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS  12

STEREO ATTRIBUTES: NONE
L22         5953 SEA FILE=REGISTRY SSS FUL L20
L28              STR
```

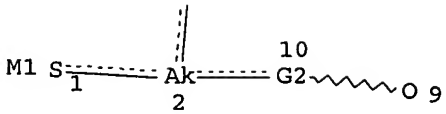
O 12 S 13







Page 1-A



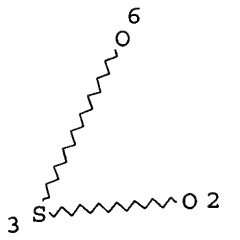
Page 2-A
VAR G1=12/13
VAR G2=4-2 4-9/5-2 5-9

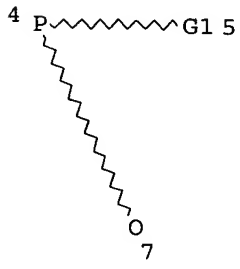
```
NODE ATTRIBUTES:
HCOUNT  IS M1     AT   1
HCOUNT  IS M1     AT  11
NSPEC   IS C      AT   1
NSPEC   IS C      AT   2
NSPEC   IS C      AT   3
NSPEC   IS C      AT   4
NSPEC   IS C      AT   5
NSPEC   IS C      AT   6
NSPEC   IS C      AT   7
NSPEC   IS C      AT   8
NSPEC   IS C      AT   9
NSPEC   IS C      AT  10
NSPEC   IS C      AT  11
CONNECT IS X3  RC AT   2
CONNECT IS E1  RC AT   9
DEFAULT MLEVEL IS ATOM
MLEVEL  IS CLASS  AT   1  2  3  4  5  7  8  9 11 12 13
DEFAULT ECLEVEL IS LIMITED

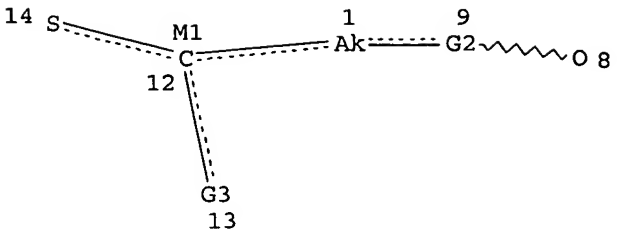
GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS  13

STEREO ATTRIBUTES: NONE
L30           69 SEA FILE=REGISTRY SUB=L22 SSS FUL L28
L31              STR
```

O 15 S 16







10 O M1

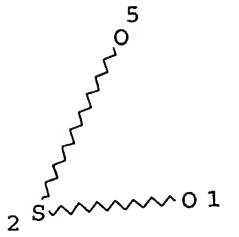
11 S M1

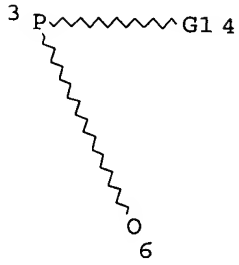
Page 2-A
VAR G1=15/16
VAR G2=3-1 3-8/4-1 4-8
VAR G3=10/11
NODE ATTRIBUTES:
HCOUNT IS M1 AT 10
HCOUNT IS M1 AT 11
HCOUNT IS M1 AT 12
NSPEC IS C AT 1
NSPEC IS C AT 2
NSPEC IS C AT 3
NSPEC IS C AT 4
NSPEC IS C AT 5
NSPEC IS C AT 6
NSPEC IS C AT 7
NSPEC IS C AT 8
NSPEC IS C AT 9
NSPEC IS C AT 10
NSPEC IS C AT 11
NSPEC IS C AT 12
NSPEC IS C AT 13
NSPEC IS C AT 14
CONNECT IS E2 RC AT 1
CONNECT IS E1 RC AT 8
CONNECT IS X2 RC AT 14
DEFAULT MLEVEL IS ATOM
MLEVEL IS CLASS AT 1 2 3 4 6 7 8 10 11 12 14 15 16
DEFAULT ECLEVEL IS LIMITED

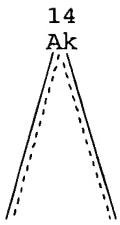
GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 16

STEREO ATTRIBUTES: NONE
L33 7 SEA FILE=REGISTRY SUB=L22 SSS FUL L31
L34 STR

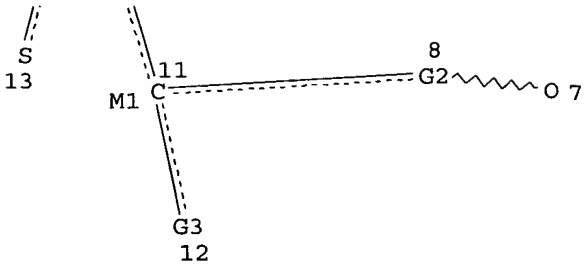
O 15 S 16







Page 1-A



9 O M1

10 S M1

Page 2-A
VAR G1=15/16
VAR G2=2-7 2-11/3-7 3-11
VAR G3=9/10
NODE ATTRIBUTES:
HCOUNT IS M1 AT 9
HCOUNT IS M1 AT 10
HCOUNT IS M1 AT 11
NSPEC IS C AT 1
NSPEC IS C AT 2
NSPEC IS C AT 3

```
NSPEC   IS C       AT   4
NSPEC   IS C       AT   5
NSPEC   IS C       AT   6
NSPEC   IS C       AT   7
NSPEC   IS C       AT   8
NSPEC   IS C       AT   9
NSPEC   IS C       AT  10
NSPEC   IS C       AT  11
NSPEC   IS C       AT  12
NSPEC   IS C       AT  13
NSPEC   IS C       AT  14
CONNECT IS E1  RC AT   7
CONNECT IS X2  RC AT  13
CONNECT IS E2  RC AT  14
DEFAULT MLEVEL IS ATOM
MLEVEL  IS CLASS  AT   1  2  3  5  6  7  9 10 11 13 14 15 16
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS  16

STEREO ATTRIBUTES: NONE
L36           34 SEA FILE=REGISTRY SUB=L22 SSS FUL L34
L47            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  ETHANESULFONIC ACID,
                 2-MERCAPTO- /CN
L50         5844 SEA FILE=REGISTRY ABB=ON  PLU=ON  L22 NOT (L5 OR L30 OR L33 OR
                 L36 OR L47)
L51         3662 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L50
L52           60 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L51 AND ((L11 OR L12) OR L15)


=> file medline
FILE 'MEDLINE' ENTERED AT 11:47:55 ON 15 JUN 2006

 FILE LAST UPDATED: 14 JUN 2006 (20060614/UP).  FILE COVERS 1950 TO DATE.

 On December 11, 2005, the 2006 MeSH terms were loaded.

 The MEDLINE reload for 2006 is now (26 Feb.) available.  For details
 on the 2006 reload, enter HELP RLOAD at an arrow prompt (=>).
 See also:

    http://www.nlm.nih.gov/mesh/
    http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html
    http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html
    http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html

 OLDMEDLINE is covered back to 1950.

 MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the
 MeSH 2006 vocabulary.

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.

=> d que nos L65
L5             1 SEA FILE=REGISTRY ABB=ON  PLU=ON  MESNA/CN
L20              STR
```

```
L22         5953 SEA FILE=REGISTRY SSS FUL L20
L28              STR
L30           69 SEA FILE=REGISTRY SUB=L22 SSS FUL L28
L31              STR
L33            7 SEA FILE=REGISTRY SUB=L22 SSS FUL L31
L34              STR
L36           34 SEA FILE=REGISTRY SUB=L22 SSS FUL L34
L47            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  ETHANESULFONIC ACID,
                 2-MERCAPTO- /CN
L50         5844 SEA FILE=REGISTRY ABB=ON  PLU=ON  L22 NOT (L5 OR L30 OR L33 OR
                 L36 OR L47)
L53           13 SEA FILE=REGISTRY ABB=ON  PLU=ON  L50 AND MEDLINE/LC
L55        51078 SEA FILE=MEDLINE ABB=ON  PLU=ON  RADIATION-PROTECTIVE AGENTS+NT
                 /CT
L62            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  MAFOSFAMIDE/CN
L63           12 SEA FILE=REGISTRY ABB=ON  PLU=ON  L53 NOT L62
L64          244 SEA FILE=MEDLINE ABB=ON  PLU=ON  L63
L65            4 SEA FILE=MEDLINE ABB=ON  PLU=ON  L64 AND L55


=> d que nos L66
L5             1 SEA FILE=REGISTRY ABB=ON  PLU=ON  MESNA/CN
L20              STR
L22         5953 SEA FILE=REGISTRY SSS FUL L20
L28              STR
L30           69 SEA FILE=REGISTRY SUB=L22 SSS FUL L28
L31              STR
L33            7 SEA FILE=REGISTRY SUB=L22 SSS FUL L31
L34              STR
L36           34 SEA FILE=REGISTRY SUB=L22 SSS FUL L34
L47            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  ETHANESULFONIC ACID,
                 2-MERCAPTO- /CN
L50         5844 SEA FILE=REGISTRY ABB=ON  PLU=ON  L22 NOT (L5 OR L30 OR L33 OR
                 L36 OR L47)
L53           13 SEA FILE=REGISTRY ABB=ON  PLU=ON  L50 AND MEDLINE/LC
L56       368763 SEA FILE=MEDLINE ABB=ON  PLU=ON  ?RADIAT?
L62            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  MAFOSFAMIDE/CN
L63           12 SEA FILE=REGISTRY ABB=ON  PLU=ON  L53 NOT L62
L64          244 SEA FILE=MEDLINE ABB=ON  PLU=ON  L63
L66            9 SEA FILE=MEDLINE ABB=ON  PLU=ON  L64 AND L56


=> d que nos L84
L5             1 SEA FILE=REGISTRY ABB=ON  PLU=ON  MESNA/CN
L20              STR
L22         5953 SEA FILE=REGISTRY SSS FUL L20
L28              STR
L30           69 SEA FILE=REGISTRY SUB=L22 SSS FUL L28
L31              STR
L33            7 SEA FILE=REGISTRY SUB=L22 SSS FUL L31
L34              STR
L36           34 SEA FILE=REGISTRY SUB=L22 SSS FUL L34
L47            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  ETHANESULFONIC ACID,
                 2-MERCAPTO- /CN
L50         5844 SEA FILE=REGISTRY ABB=ON  PLU=ON  L22 NOT (L5 OR L30 OR L33 OR
                 L36 OR L47)
L53           13 SEA FILE=REGISTRY ABB=ON  PLU=ON  L50 AND MEDLINE/LC
L55        51078 SEA FILE=MEDLINE ABB=ON  PLU=ON  RADIATION-PROTECTIVE AGENTS+NT
                 /CT
L56       368763 SEA FILE=MEDLINE ABB=ON  PLU=ON  ?RADIAT?
```

```
L60        32292 SEA FILE=MEDLINE ABB=ON  PLU=ON  RADIATION, IONIZING+NT/CT
L79            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  L53 AND S=6
L80            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  L53 AND C3H8O3S2/MF
L81            2 SEA FILE=REGISTRY ABB=ON  PLU=ON  (L79 OR L80)
L82              SEL  PLU=ON  L81 1- CHEM :       4 TERMS
L83          219 SEA FILE=MEDLINE ABB=ON  PLU=ON  L82
L84            6 SEA FILE=MEDLINE ABB=ON  PLU=ON  (L55 OR L56 OR L60) AND L83

=> s L65 or L66 or L84
L95           17 L65 OR L66 OR L84

=> file embase
FILE 'EMBASE' ENTERED AT 11:48:51 ON 15 JUN 2006
Copyright (c) 2006 Elsevier B.V. All rights reserved.

 FILE COVERS 1974 TO 15 Jun 2006 (20060615/ED)

 EMBASE has been reloaded. Enter HELP RLOAD for details.

 EMBASE is now updated daily.  SDI frequency remains weekly (default)
 and biweekly.

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.

=> d que nos L78
L5             1 SEA FILE=REGISTRY ABB=ON  PLU=ON  MESNA/CN
L20              STR
L22         5953 SEA FILE=REGISTRY SSS FUL L20
L28              STR
L30           69 SEA FILE=REGISTRY SUB=L22 SSS FUL L28
L31              STR
L33            7 SEA FILE=REGISTRY SUB=L22 SSS FUL L31
L34              STR
L36           34 SEA FILE=REGISTRY SUB=L22 SSS FUL L34
L47            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  ETHANESULFONIC ACID,
                 2-MERCAPTO- /CN
L50         5844 SEA FILE=REGISTRY ABB=ON  PLU=ON  L22 NOT (L5 OR L30 OR L33 OR
                 L36 OR L47)
L69            7 SEA FILE=REGISTRY ABB=ON  PLU=ON  L50 AND EMBASE/LC
L70            4 SEA FILE=REGISTRY ABB=ON  PLU=ON  L69 AND RSD/FA
L71            3 SEA FILE=REGISTRY ABB=ON  PLU=ON  L69 NOT L70
L74         6762 SEA FILE=EMBASE ABB=ON  PLU=ON  RADIOPROTECTIVE AGENT+ALL/CT
L76              SEL  PLU=ON  L71 1- CHEM :       17 TERMS
L77          352 SEA FILE=EMBASE ABB=ON  PLU=ON  L76
L78           17 SEA FILE=EMBASE ABB=ON  PLU=ON  L77 AND L74

=> file biosis
FILE 'BIOSIS' ENTERED AT 11:49:07 ON 15 JUN 2006
Copyright (c) 2006 The Thomson Corporation

FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 14 June 2006 (20060614/ED)
```

```
=> d que nos L91
L5            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  MESNA/CN
L20             STR
L22        5953 SEA FILE=REGISTRY SSS FUL L20
L28             STR
L30          69 SEA FILE=REGISTRY SUB=L22 SSS FUL L28
L31             STR
L33           7 SEA FILE=REGISTRY SUB=L22 SSS FUL L31
L34             STR
L36          34 SEA FILE=REGISTRY SUB=L22 SSS FUL L34
L47           1 SEA FILE=REGISTRY ABB=ON  PLU=ON  ETHANESULFONIC ACID,
                2-MERCAPTO- /CN
L50        5844 SEA FILE=REGISTRY ABB=ON  PLU=ON  L22 NOT (L5 OR L30 OR L33 OR
                L36 OR L47)
L85          14 SEA FILE=REGISTRY ABB=ON  PLU=ON  L50 AND BIOSIS/LC
L86           8 SEA FILE=REGISTRY ABB=ON  PLU=ON  L85 AND RSD/FA
L87           6 SEA FILE=REGISTRY ABB=ON  PLU=ON  L85 NOT L86
L88             SEL  PLU=ON  L87 1- CHEM :      25 TERMS
L89         523 SEA FILE=BIOSIS ABB=ON  PLU=ON  L88
L90      462658 SEA FILE=BIOSIS ABB=ON  PLU=ON  ?RADIAT?
L91           2 SEA FILE=BIOSIS ABB=ON  PLU=ON  L89 AND L90


=> d que nos L94
L5            1 SEA FILE=REGISTRY ABB=ON  PLU=ON  MESNA/CN
L20             STR
L22        5953 SEA FILE=REGISTRY SSS FUL L20
L28             STR
L30          69 SEA FILE=REGISTRY SUB=L22 SSS FUL L28
L31             STR
L33           7 SEA FILE=REGISTRY SUB=L22 SSS FUL L31
L34             STR
L36          34 SEA FILE=REGISTRY SUB=L22 SSS FUL L34
L47           1 SEA FILE=REGISTRY ABB=ON  PLU=ON  ETHANESULFONIC ACID,
                2-MERCAPTO- /CN
L50        5844 SEA FILE=REGISTRY ABB=ON  PLU=ON  L22 NOT (L5 OR L30 OR L33 OR
                L36 OR L47)
L85          14 SEA FILE=REGISTRY ABB=ON  PLU=ON  L50 AND BIOSIS/LC
L86           8 SEA FILE=REGISTRY ABB=ON  PLU=ON  L85 AND RSD/FA
L87           6 SEA FILE=REGISTRY ABB=ON  PLU=ON  L85 NOT L86
L88             SEL  PLU=ON  L87 1- CHEM :      25 TERMS
L89         523 SEA FILE=BIOSIS ABB=ON  PLU=ON  L88
L92       70394 SEA FILE=BIOSIS ABB=ON  PLU=ON  ?RADIOTHERAP?
L93        8062 SEA FILE=BIOSIS ABB=ON  PLU=ON  ?RADIOPROTECT?
L94           0 SEA FILE=BIOSIS ABB=ON  PLU=ON  L89 AND (L92 OR L93)


=> s L91 or L94
L96           2 L91 OR L94

=> dup rem L52 L95 L78 L96
FILE 'HCAPLUS' ENTERED AT 11:49:48 ON 15 JUN 2006
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2006 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'MEDLINE' ENTERED AT 11:49:48 ON 15 JUN 2006

FILE 'EMBASE' ENTERED AT 11:49:48 ON 15 JUN 2006
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FILE 'BIOSIS' ENTERED AT 11:49:48 ON 15 JUN 2006

PROCESSING COMPLETED FOR L52
PROCESSING COMPLETED FOR L95
PROCESSING COMPLETED FOR L78
PROCESSING COMPLETED FOR L96
L97 90 DUP REM L52 L95 L78 L96 (6 DUPLICATES REMOVED)
ANSWERS '1-60' FROM FILE HCAPLUS
ANSWERS '61-74' FROM FILE MEDLINE
ANSWERS '75-89' FROM FILE EMBASE
ANSWER '90' FROM FILE BIOSIS

=> d ibib abs hitind hitstr L97 1-60; d iall L97 61-90

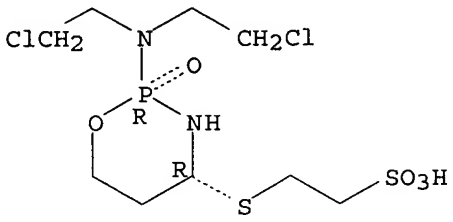
L97 ANSWER 1 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 4
ACCESSION NUMBER: 1991:670160 HCAPLUS
DOCUMENT NUMBER: 115:270160
TITLE: Use and mechanism of action of AS101 in protecting bone marrow colony forming units-granulocyte-macrophage following purging with ASTA-Z 7557
AUTHOR(S): Kalechman, Yona; Barkai, Iris Sotnik; Albeck, Michael; Horwith, Gary; Sehagl, Suren N.; Sredni, Benjamin
CORPORATE SOURCE: Dep. Life Sci., Bar Ilan Univ., Ramat Gan, 59200, Israel
SOURCE: Cancer Research (1991), 51(20), 5614-20
CODEN: CNREA8; ISSN: 0008-5472
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Ammonium trichloro(dioxoethylene-O,O')tellurate (AS101) has been shown previously to provide **radioprotective** effects when given to mice 24 h prior to irradiation and to protect mice from lethal and sublethal doses of cyclophosphamide (CTX). In this study, the ability of AS101 to protect mice bone marrow colony forming units-granulocyte-macrophage treated in vitro with various doses of ASTA-Z 7557 (I) a potent derivative of CTX were examined Prior incubation with I protects colony forming units-granulocyte-macrophage from toxic effects of I. This protection can also be conferred by injection of mice with AS101 prior to incubation of their bone marrow in vitro with I. Prior incubation with AS101 was shown not to protect K562 leukemic cells or HL-60 cells from the toxic effects of I. AS101 protection from the toxic effects of I in vitro and CTX in vivo can be partially ascribed to increased aldehyde dehydrogenase (ALDH) activity induced by AS101. This was shown directly by measuring cellular ALDH activity and indirectly by measuring the toxicity of I and CTX in the presence of cyanamide, an inhibitor of ALDH. AS101 also protects spleen cells from the toxic effects of 5-fluorouracil, probably through a different mechanism. These properties of AS101 make it a useful candidate for increasing the qual. potential of bone marrow used for autologous transplantation after purging with I. In addition, the results suggest an increase in ALDH activity by AS101 as one of the mechanisms of protection from the toxic effects of I and CTX. However, the chemoprotectiveness of AS101 was not restricted to cyclophosphamide, since as shown in this study, AS101 helped by other mechanisms to reconstitute the number of spleen cells after 5-fluorouracil treatment.
CC 1-6 (Pharmacology)
IT 50-18-0, Cyclophosphamide **84210-80-0**, ASTA-Z 7557
RL: BIOL (Biological study)
(leukemia purging from bone marrow autotransplant by, AS101 protection in)
IT **84210-80-0**, ASTA-Z 7557

RL: BIOL (Biological study)
(leukemia purging from bone marrow autotransplant by, AS101 protection in)
RN 84210-80-0 HCAPLUS
CN Ethanesulfonic acid, 2-[[(2R,4R)-2-[bis(2-chloroethyl)amino]tetrahydro-2-oxido-2H-1,3,2-oxazaphosphorin-4-yl]thio]-, rel-, compd. with cyclohexanamine (1:1) (9CI) (CA INDEX NAME)

CM 1

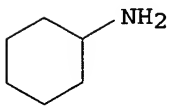
CRN 88859-04-5
CMF C9 H19 Cl2 N2 O5 P S2

Relative stereochemistry.



CM 2

CRN 108-91-8
CMF C6 H13 N



L97 ANSWER 2 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 5
ACCESSION NUMBER: 1987:568357 HCAPLUS
DOCUMENT NUMBER: 107:168357
TITLE: Preferential differentiation of murine CFU-S toward granulopoiesis and megakaryocytopoiesis after in vitro incubation of bone marrow with ASTA-Z 7557
AUTHOR(S): Sainteny, Francoise; Lopez, Manuel; Mary, Jean Yves; Frindel, Emilia
CORPORATE SOURCE: Cell. Kinet. Res. Unit, Gustave-Roussy Inst., Villejuif, Fr.
SOURCE: Experimental Hematology (New York, NY, United States) (1987), 15(6), 631-5
CODEN: EXHMA6; ISSN: 0301-472X
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In vitro effect of ASTA-Z 7557 on the qual. aspects of murine CFU-S (pluripotent stem cells) differentiation (as assessed by the histol. nature of day-9 colonies generated in the spleen of **irradiated** mice by bone marrow exposed to the drug at concns. ranging from 0 to 150 μg/mL) was investigated. The proportion of erythrocytic colonies declined linearly with the logarithm of the dose (a 22% decrease per log),

whereas the granulocytic and megakaryocytic colony proportions increased linearly (a 10% increase per log for both cell lineages). This suggests a preferential channeling of CFU-S differentiation toward granulopoietic and megakaryocytic cell lineages as a consequence of the in vitro chemotherapy, and supports the hypothesis that some alteration of the qual. potential of CFU-S to diffeentiate after in vitro purging of bone marrow with ASTA-Z 7557 takes place prior to autologous bone marrow transplantation.

CC 1-6 (Pharmacology)

IT **84210-80-0**, ASTA-Z 7557
RL: BIOL (Biological study)
(bone marrow purging in vitro with, stem cell differentiation response to)

IT **84210-80-0**, ASTA-Z 7557
RL: BIOL (Biological study)
(bone marrow purging in vitro with, stem cell differentiation response to)

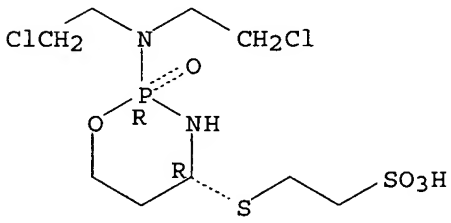
RN 84210-80-0 HCAPLUS

CN Ethanesulfonic acid, 2-[[(2R,4R)-2-[bis(2-chloroethyl)amino]tetrahydro-2-oxido-2H-1,3,2-oxazaphosphorin-4-yl]thio]-, rel-, compd. with cyclohexanamine (1:1) (9CI) (CA INDEX NAME)

CM 1

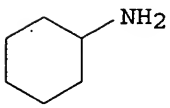
CRN 88859-04-5
CMF C9 H19 Cl2 N2 O5 P S2

Relative stereochemistry.



CM 2

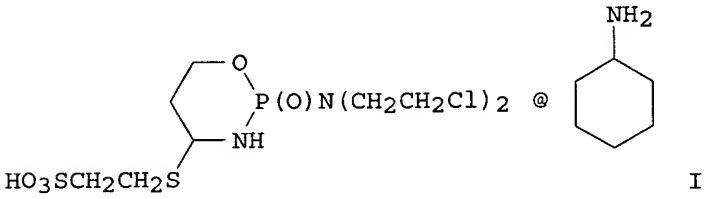
CRN 108-91-8
CMF C6 H13 N



L97 ANSWER 3 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 6

ACCESSION NUMBER: 1984:603988 HCAPLUS
DOCUMENT NUMBER: 101:203988
TITLE: No preferential sensitivity of clonogenic AML cells to ASTA-Z-7557
AUTHOR(S): Kluin-Nelemans, Hanneke C.; Martens, Anton C. M.; Loewenberg, Bob; Hagenbeek, Anton

CORPORATE SOURCE: Dep. Hematol., Dr. Daniel den Hoed Cancer Cent., Rotterdam, 3008 AE, Neth.
SOURCE: Leukemia Research (1984), 8(4), 723-8
CODEN: LEREDD; ISSN: 0145-2126
DOCUMENT TYPE: Journal
LANGUAGE: English
GI



I

AB ASTA-Z-7557 (I) [**84210-80-0**], an in vitro active metabolite of cyclophosphamide, has recently been introduced to purge autologous bone marrow grafts of patients with acute myeloid leukemia (AML). The rationale of this approach assumes a relatively higher sensitivity of leukemic cells to the drug than of normal marrow precursors. The sensitivity to ASTA-Z-7557 of normal bone marrow progenitors (GM-CFC and BFU-E) and clonogenic leukemic cells (L-CFC) was compared. Normal bone marrow cells and purified leukemic blast cells were exposed to varying concns. of the drug. Concentration-response relationships did not indicate a selective cytotoxic susceptibility of L-CFC to ASTA-Z-7557. The recovery of bone marrow precursors following exposure to ASTA-Z-7557 depended on the cell concentration during exposure and was higher for 2 + 107 cells/mL than for 1 + 106/mL. To mimic minimal residual leukemia, cell mixts. of 95% **irradiated** normal bone marrow cells with 5% leukemic blast cells were exposed to ASTA-Z-7557. In this mixture killing of L-CFC was largely decreased. These data suggest that in vitro incubation of autologous bone marrow grafts of patients with minimal residual leukemia with ASTA-Z-7557 might not offer a therapeutic advantage.

CC 1-6 (Pharmacology)

IT **84210-80-0**
RL: BIOL (Biological study)
(clonogenic acute myeloid leukemia cells of humans response to)

IT **84210-80-0**
RL: BIOL (Biological study)
(clonogenic acute myeloid leukemia cells of humans response to)

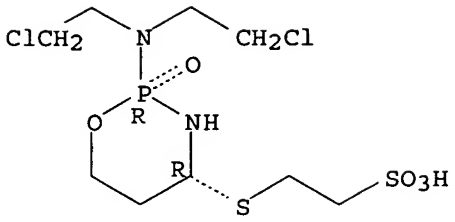
RN 84210-80-0 HCAPLUS

CN Ethanesulfonic acid, 2-[[(2R,4R)-2-[bis(2-chloroethyl)amino]tetrahydro-2-oxido-2H-1,3,2-oxazaphosphorin-4-yl]thio]-, rel-, compd. with cyclohexanamine (1:1) (9CI) (CA INDEX NAME)

CM 1

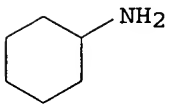
CRN 88859-04-5
CMF C9 H19 Cl2 N2 O5 P S2

Relative stereochemistry.



CM 2

CRN 108-91-8
CMF C6 H13 N



L97 ANSWER 4 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2006:273690 HCAPLUS
DOCUMENT NUMBER: 144:305181
TITLE: Method of treatment for or protection against lymphedema
INVENTOR(S): Hausheer, Frederick H.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 5 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

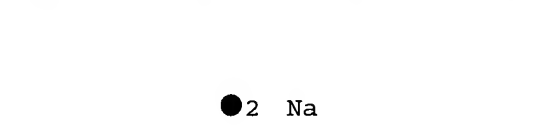
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2006063742	A1	20060323	US 2004-945754	20040921
WO 2006034325	A2	20060330	WO 2005-US33771	20050921

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: US 2004-945754 A 20040921
OTHER SOURCE(S): MARPAT 144:305181
AB A method of reducing the risks of lymphedema, particularly secondary lymphedema associated with surgery or **radiotherapy** is disclosed. The method of this invention includes administering to a patient at risk

of developing lymphedema effective amts. of specific sulfur-containing drug agents according to (R1SR2), wherein R1 is hydrogen, lower alkyl or -S-R2-R3; R2 is lower alkylene, optionally substituted by one or more hydroxy, alkoxy, mercapto, nitro or amino moieties for a corresponding hydrogen atom; and R3 is sulfonate or phosphonate; and pharmaceutically acceptable salts thereof.

INCL 514114000; 514553000; 514126000
CC 1-12 (Pharmacology)
Section cross-reference(s): 8, 63
ST lymphedema sulfur drug surgery **radiotherapy**
IT Human
Lymph node, disease
Radiotherapy
Surgery
(method of treatment for or protection against lymphedema)
IT 7704-34-9D, Sulfur, -containing drugs **16208-51-8** 19767-45-4
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(method of treatment for or protection against lymphedema)
IT **16208-51-8**
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(method of treatment for or protection against lymphedema)
RN 16208-51-8 HCAPLUS
CN Ethanesulfonic acid, 2,2'-dithiobis-, disodium salt (9CI) (CA INDEX NAME)



L97 ANSWER 5 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:1314342 HCAPLUS
DOCUMENT NUMBER: 144:57543
TITLE: Combination product comprising anastrozole and a dual prenyl transferase inhibitor
INVENTOR(S): Stephens, Trevor Charles
PATENT ASSIGNEE(S): Astrazeneca AB, Swed.; Astrazeneca Uk Limited
SOURCE: PCT Int. Appl., 23 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005117864	A1	20051215	WO 2005-GB2079	20050525

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT,

RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: GB 2004-12004 A 20040528
GB 2004-17722 A 20040810

AB The invention concerns a combination therapeutic product comprising anastrozole and a dual prenyl transferase inhibitor that inhibits both farnesyl transferase and geranylgeranyl transferase-1 for use simultaneously, sequentially or sep. in the treatment or prophylaxis of breast cancer.

IC ICM A61K031-4196
ICS A61K031-4439; A61K031-663; A61K035-00

CC 63-6 (Pharmaceuticals)
Section cross-reference(s): 1, 8

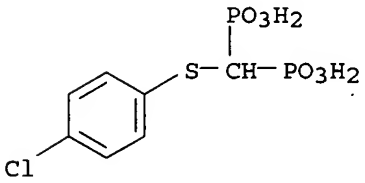
IT Antitumor agents
Combination chemotherapy
Human
Mammary gland, neoplasm
Radiotherapy
(combination product comprising anastrozole and a dual prenyl transferase inhibitor)

IT 2809-21-4 40391-99-9 66376-36-1, Alendronate **89987-06-4**, Tiludronate 105462-24-6 120511-73-1, Anastrozole 345915-10-8, AZD 3409
RL: PAC (Pharmacological activity); PEP (Physical, engineering or chemical process); PYP (Physical process); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(combination product comprising anastrozole and a dual prenyl transferase inhibitor)

IT **89987-06-4**, Tiludronate
RL: PAC (Pharmacological activity); PEP (Physical, engineering or chemical process); PYP (Physical process); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(combination product comprising anastrozole and a dual prenyl transferase inhibitor)

RN 89987-06-4 HCAPLUS

CN Phosphonic acid, [[(4-chlorophenyl)thio]methylene]bis- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L97 ANSWER 6 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:1290072 HCAPLUS
DOCUMENT NUMBER: 144:46998
TITLE: The X-ray crystal structure of BRCA1 tandem BRCT repeat and BACH1 phosphopeptide complex and methods and compositions for antitumor drug design
INVENTOR(S): Yaffe, Michael B.; Clapperton, Julie A.; Manke, Isaac A.; Lowery, Drew M.; Ho, Timmy; Haire, Lesley F.; Smerdon, Stephen J.

PATENT ASSIGNEE(S): Massachusetts Institute of Technology, USA
SOURCE: PCT Int. Appl., 360 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005115454	A2	20051208	WO 2005-US15981	20050509

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SM, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2004-569131P P 20040507

AB The present invention relates to compds. (e.g., peptidomimetics and non-peptides) that treat, prevent or stabilize cellular proliferative disorders and methods of treating, preventing, or stabilizing such disorders. The invention also provides three-dimensional structures of a BRCT domain-BACH1 phosphopeptide complex.

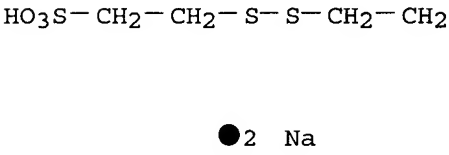
IC ICM A61K039-395
CC 6-3 (General Biochemistry)
Section cross-reference(s): 1, 3, 13
IT **Radiotherapy**
(for cancer treatment; X-ray crystal structure of BRCA1 tandem BRCT repeat and BACH1 phosphopeptide complex and methods and compns. for antitumor drug design)
IT 50-02-2, Dexamethasone 50-07-7, Mitomycin C 50-18-0, Cyclophosphamide 50-24-8, Prednisolone 50-44-2, 6-Mercaptopurine 50-76-0, Dactinomycin 50-91-9, Floxuridine 51-21-8, 5-Fluorouracil 51-45-6, Histamine, biological studies 51-75-2, Mechlorethamine 52-24-4, Thiotepa 53-03-2, Prednisone 53-19-0, Mitotane 55-98-1, Busulfan 56-53-1, Diethylstilbestrol 57-22-7, Vincristine 57-63-6, Ethinyl estradiol 58-18-4, Methyltestosterone 58-22-0, Testosterone 59-05-2, Methotrexate 64-86-8, Colchicine 72-03-7, Propionate, biological studies 76-43-7, Fluoxymesterone 79-09-4, Propanoic acid, biological studies 83-43-2, Methylprednisolone 125-84-8, Aminoglutethimide 127-07-1, Hydroxyurea 129-56-6, Anthra[1,9-cd]pyrazol-6(2H)-one 147-94-4, Cytarabin 148-82-3, Melphalan 154-42-7, 6-Thioguanine 154-93-8, Carmustine 302-79-4, trans-Retinoic acid 305-03-3, Chlorambucil 320-67-2, Azacytidine 362-07-2, 2-Methoxyestradiol 520-85-4, Medroxyprogesterone 536-59-4, Perillyl alcohol 548-04-9, Hypericin 566-48-3, Formestane 569-57-3, Chlortrianizen 616-91-1, N-Acetylcysteine 630-56-8, Hydroxyprogesterone caproate 645-05-6, Hexamethylmelamine 646-08-2, β-Alethine 671-16-9, Procarbazine 801-52-5, Porfiromycin 865-21-4, Vinblastine 2353-33-5, Decitabine 3432-99-3, CoFactor 3562-63-8, Megestrol 3778-73-2, Ifosfamide 4105-38-8, 2',3'-5'-Triacetyluridine 4291-63-8, 2-Chlorodeoxyadenosine 4342-03-4, Dacarbazine 4891-15-0, Estramustine phosphate 5300-03-8, Alitretinoin 5825-87-6, 3CPA 7440-06-4D, Platinum, derivs. 9032-75-1, PG2 9041-93-4, Bleomycin sulfate 10212-20-1,

2'-Fluoro-2'-deoxycytidine 10540-29-1, Tamoxifen 11003-32-0, Bleomycin A 11003-33-1, Bleomycin B 13010-47-4, Lomustine 13311-84-7, Flutamide 13909-09-6, Semustine 15663-27-1, Cisplatin 15866-90-7, CMT-3 **16208-51-8**, BNP-7787 18378-89-7, Plicamycin 18883-66-4, Streptozocin 19916-73-5, O6 Benzylguanine 20830-81-3, Daunorubicin 21679-14-1, Fludarabine 23214-92-8, Doxorubicin 24584-09-6, Dexrazoxane 26833-87-4, Ceflatonin 27314-97-2, Tirapazamine 29767-20-2, Teniposide 33069-62-4, Paclitaxel 33069-62-4D, Paclitaxel, PED-conjugated 33419-42-0, Etoposide 37364-66-2, Bleomycinic acid 41575-94-4, Carboplatinum 41941-56-4, Tocladesine 51264-14-3, Amsacrine 51543-40-9, (R)-Flurbiprofen 52128-35-5, Trimetrexate 53643-48-4, Vindesine 53714-56-0, Leuprolide 53910-25-1, Deoxycoformycin 54083-22-6, Rubidazone 56124-62-0, Valrubicin 56420-45-2, Epirubicin 56509-01-4, Immunol 58957-92-9, Idarubicin 59973-80-7, Exisulind 60084-10-8, Tiazofurin 61825-94-3, Oxaliplatin 62928-11-4, Iproplatin 63521-85-7, 4'-Deoxydoxorubicin 63612-50-0, Nilutamide 65223-78-1, 5-Ethynylcytidine 65271-80-9, Mitoxantrone 65646-68-6, Fenretinide 65807-02-5, Goserelin 69408-81-7, Amonafide 69839-83-4, Didox 70052-12-9, Eflornithine 71486-22-1, Vinorelbine 72496-41-4, Therarubicin 74790-08-2, Spiroplatin 75706-12-6, Leflunomide 81267-65-4, Phenoxodiol 83150-76-9, Octreotide 83314-01-6, Bryostatin-1 84692-91-1, Arglabin 85622-93-1, Temozolomide 86639-52-3, 7-Ethyl-10-hydroxycamptothecin 88254-07-3, MRA-CN 88303-60-0, Losoxantrone **88859-04-5**, Mafosfamide 89778-26-7, Toremifene 90357-06-5, Bicalutamide 90996-54-6, Rhizoxin 91421-42-0, Rubitecan 91441-23-5, Oxantrazole 93908-02-2D, Rebeccamycin, analog 95058-81-4, Gemcitabine 96301-34-7, Atamestane 97068-30-9, Elsamitrucin 97682-44-5, Irinotecan 98774-23-3, Tesmilifene 107868-30-4, Exemestane 108560-70-9, Gallium maltolate 110230-98-3, Talaporfin 110417-88-4, Dolastatin-10- 111358-88-4, CEP-701 112522-64-2, Tacedinaline 112809-51-5, Letrozole 112887-68-0, Tomudex 114560-48-4, Apaziquone 114899-77-3, Trabectedin 114977-28-5, Docetaxel 117048-59-6, Combretastatin A4 119804-96-5, DMDC 120511-73-1, Anastrozole 120685-11-2, PKC412 122110-53-6, Pivaloyloxymethyl butyrate 122332-18-7, Mivobulin 123318-82-1, Clofarabine 123948-87-8, Topotecan 125313-92-0, Ro-31-7453 126411-13-0, Apomine 129580-63-8, Satraplatin 130306-02-4, Tezacitabine 131179-95-8, Efaproxiral 131384-38-8, Farnesyltransferase 132173-07-0, SR 31747 132682-98-5, Glufosfamide 134404-52-7, Seocalcitol 135558-11-1, Lobaplatin 136381-85-6, SR-27897 137219-37-5, Aplidine 137281-23-3, Pemetrexed 140917-67-5, Azonafide 141430-65-1, E7010 141977-79-9, SM-11355 143621-35-6, Triapine 144510-96-3, Pixantrone 146426-40-6, Alvocidib 147149-76-6, Nolatrexed 148717-90-2, Squalamine 148869-05-0, YM-511 149204-42-2, Kahalalide F 149606-27-9, Auristatin PE 149647-78-9, SAHA 149682-77-9, PT-100 149838-23-3, Doranidazole 150091-68-2, Quinamed 152044-54-7, Epothilone B 152459-95-5, Imatinib 153537-73-6, ZD-9331 154039-60-8, Marimastat 154361-50-9, Capecitabine 156090-18-5, BBR-3576 156177-59-2, CEP-751 157078-48-3, Isohomohalichondrin-B 158440-71-2, Irofulven 158681-49-3, MS-209 159776-69-9, Cemadotin 160237-25-2, BMS 184476 162635-04-3, CCI-779 162652-95-1, Vinflunine 165668-41-7, Indisulam 167465-36-3, Zosuquidar trihydrochloride 169317-77-5, MEN-10755 169869-90-3, Exatecan mesylate 172481-83-3, BMS 188797 172903-00-3, BBR-3464 173937-91-2, Atrasentan 174254-13-8, Biricodar dicitrate 174402-32-5, J-107088- 174634-09-4, TAS-103 174722-31-7, Rituximab 178600-20-9, LGD-1550 179324-69-7, Bortezomib 180064-38-4, Minodronic acid 180288-69-1, Trastuzumab 181630-15-9, ZD-0473 182133-25-1, Arzoxifene 183133-96-2, TXD 258 183321-74-6, Erlotinib 184475-35-2, ZD1839 185077-23-0, PI 88 186256-67-7, Cryptophycin 52 186348-23-2, IDN 5109 186497-07-4, ZD-4054 187724-61-4, PKI166

188968-51-6, Cilengitide 191732-72-6, Revimid 192185-72-1, Tipifarnib
192573-38-9, RPR 109881A 193275-84-2, Sarasar 195533-53-0, T 138067
195612-80-7, Galarubicin 196488-72-9, Ranpirnase 199796-52-6,
Taxoprexin 200484-11-3, CHS-828 203258-60-0, Brostallicin
203923-89-1, BNP-1350 204005-46-9, SU5416 204205-90-3, D24851
204318-14-9, Edotreotide
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(X-ray crystal structure of BRCA1 tandem BRCT repeat and BACH1 phosphopeptide complex and methods and compns. for antitumor drug design)

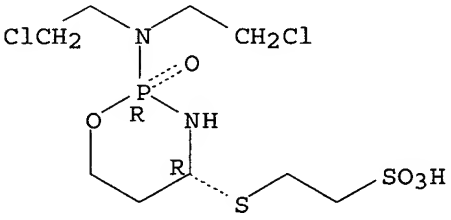
IT **16208-51-8**, BNP-7787 **88859-04-5**, Mafosfamide
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(X-ray crystal structure of BRCA1 tandem BRCT repeat and BACH1 phosphopeptide complex and methods and compns. for antitumor drug design)

RN 16208-51-8 HCAPLUS
CN Ethanesulfonic acid, 2,2'-dithiobis-, disodium salt (9CI) (CA INDEX NAME)



RN 88859-04-5 HCAPLUS
CN Ethanesulfonic acid, 2-[[(2R,4R)-2-[bis(2-chloroethyl)amino]tetrahydro-2-oxido-2H-1,3,2-oxazaphosphorin-4-yl]thio]-, rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.



L97 ANSWER 7 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:696762 HCAPLUS
DOCUMENT NUMBER: 143:187463
TITLE: M-CSF muteins for prevention and treatment of bone metastases
INVENTOR(S): Zimmerman, Deborah Lee; Harrowe, Gregory Martin; Liu, Cheng; Koths, Kirston; Kavanaugh, William Michael; Long, Li
PATENT ASSIGNEE(S): Chiron Corporation, USA
SOURCE: PCT Int. Appl., 79 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2005070447	A2	20050804	WO 2005-US1630	20050121
WO 2005070447	A3	20051208		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, SM

RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 2004-537985P P 20040121

AB Macrophage colony-stimulating factor (M-CSF) muteins are provided, along with pharmaceutical compns. containing a M-CSF mutein, kits containing a pharmaceutical composition, methods of preventing and treating bone metastases in a subject afflicted with metastatic cancer, and methods of screening for M-CSF muteins.

IC ICM A61K038-00

CC 2-10 (Mammalian Hormones)
Section cross-reference(s): 3, 8, 15, 63

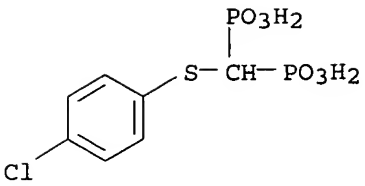
IT Animal tissue culture
Antitumor agents
Drug screening
Human
Molecular association
Mutation
Protein sequences
Radiotherapy
Surgery
cDNA sequences
(M-CSF muteins for prevention and treatment of bone metastases)

IT 2809-21-4 10596-23-3 40391-99-9 66376-36-1, Alendronate **89987-06-4**, Tiludronate 114084-78-5, Ibandronate 143011-72-7, Gcsf
RL: PAC (Pharmacological activity); PEP (Physical, engineering or chemical process); PYP (Physical process); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(M-CSF muteins for prevention and treatment of bone metastases)

IT **89987-06-4**, Tiludronate
RL: PAC (Pharmacological activity); PEP (Physical, engineering or chemical process); PYP (Physical process); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(M-CSF muteins for prevention and treatment of bone metastases)

RN 89987-06-4 HCAPLUS

CN Phosphonic acid, [[(4-chlorophenyl)thio]methylene]bis- (9CI) (CA INDEX NAME)



L97 ANSWER 8 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:1311702 HCAPLUS
DOCUMENT NUMBER: 144:57525
TITLE: Coated vaginal devices for vaginal delivery of therapeutically effective and/or health-promoting agents
INVENTOR(S): Wilson, Michelle; Desai, Kishorkumar J.; Pauletti, Giovanni M.; Antoon, Mitchell K.; Clendening, Chris E.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 40 pp., Cont.-in-part of U.S. Ser. No. 126,863
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 11
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2005276836	A1	20051215	US 2005-180076	20050712
US 6197327	B1	20010306	US 1998-79897	19980515
US 6086909	A	20000711	US 1999-249963	19990212
US 6572874	B1	20030603	US 2000-626025	20000727
NZ 508130	A	20020301	NZ 2000-508130	20001113
AU 765269	B2	20030911	AU 2001-54192	20010703
US 2003049302	A1	20030313	US 2002-226667	20020821
US 6982091	B2	20060103		
US 2004005345	A1	20040108	US 2003-349029	20030122
US 6905701	B2	20050614		
US 2004043071	A1	20040304	US 2003-600849	20030620
US 2005249774	A1	20051110	US 2005-126863	20050510
US 2006002966	A1	20060105	US 2005-208209	20050818

PRIORITY APPLN. INFO.:

US 1997-49325P	P	19970611
US 1998-79897	A2	19980515
US 1999-249963	A2	19990212
US 2000-626025	A2	20000727
US 2002-226667	A2	20020821
US 2003-349029	A2	20030122
US 2003-600849	A2	20030620
US 2004-587454P	P	20040712
US 2005-126863	A2	20050510
AU 1998-76976	A3	19980610
NZ 1998-502120	A1	19980610
US 1999-146218P	P	19990728
US 2001-315877P	P	20010829
US 2002-390748P	P	20020621

AB Disclosed is a vaginal device for delivering therapeutical and/or health-promoting agents. The vaginal device partly or completely coated by, covered by or combined with a coating or covering comprising a film,

foam, strip, cap, cup or particles. The coating of the device comprises a mucoadhesive composition comprising a therapeutical and/or health-promoting agent. For example, sumatriptan vaginal suppository were prepared from Suppocire AS2X, hydroxypropyl Me cellulose as a mucoadhesive agent, and Transcutol as a permeation enhancer.

IC ICM A61F013-00
INCL 424422000
CC 63-6 (Pharmaceuticals)
IT AIDS (disease)
Aloe barbadensis
Angelica sinensis
Anti-AIDS agents
Antimicrobial agents
Antioxidants
Antitumor agents
Areca catechu
Black cohosh
Calcium channel blockers
Calendula
Capsicum
Chamomile
Human
Hypericum perforatum
Lavandula
Melissa officinalis
Oenothera
Permeation enhancers
Potassium channel blockers
Probiotics
Rhododendron
Rosmarinus officinalis
Surfactants
Symphytum
Trigonella foenum-graecum
Vasodilators
Vigna **radiata**
Vitex agnus-castus
Witch hazel
(coated vaginal devices for vaginal delivery of therapeutically effective and/or health-promoting agents)

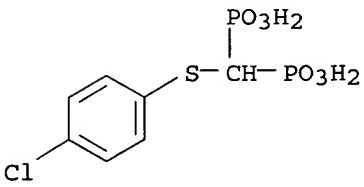
IT 69655-05-6, Didanosine 70458-96-7, Norfloxacin 71048-87-8, Levonantradol 71125-38-7, Meloxicam 72479-26-6, Fenticonazole 72509-76-3, Felodipine 73573-87-2, Formoterol 74103-06-3, Ketorolac 74103-07-4, Ketorolac tromethamine 74191-85-8, Doxazosin 74545-79-2, Aloeresin A 75088-80-1, Manoalide 75330-75-5, Lovastatin 75695-93-1, Isradipine 76584-70-8, Divalproex sodium 79350-37-1, Cefixime 79778-41-9, Neridronate 80210-62-4, Cefpodoxime 80937-31-1, Flosulide 82410-32-0, Ganciclovir 83905-01-5, Azithromycin 83991-25-7, Ambasilide 84625-61-6, Itraconazole 85622-93-1, Temozolomide 85721-33-1, Ciprofloxacin 86386-73-4, Fluconazole 88150-42-9, Amlodipine 89365-50-4, Salmeterol 89565-68-4, Tropisetron 89778-26-7, Toremifene **89987-06-4**, Tiludronate 90357-06-5, Bicalutamide 90961-53-8, Tedisamil 91714-94-2, Bromfenac 92665-29-7, Cefprozil 95058-81-4, Gemcitabine 95298-47-8 95751-30-7, Charybdotoxin 97240-79-4, Topiramate 97682-44-5, Irinotecan 98079-51-7, Lomefloxacin 98225-48-0, Brevetoxin 99614-02-5, Ondansetron 101526-83-4, Sematilide 103628-46-2, Sumatriptan 104227-87-4, Famciclovir 105462-24-6 107868-30-4, Exemestane 110042-95-0, Acemannan 112455-84-2, Papuamine 112809-51-5, Letrozole 114084-78-5, Ibandronate 114977-28-5, Docetaxel 115256-11-6,

Dofetilide 115956-12-2, Dolasetron 118072-93-8, Zoledronate 120511-73-1, Anastrozole 121368-58-9, Olpadronate 121679-13-8, Naratriptan 122647-31-8, Ibutilide 123948-87-8, Topotecan 127779-20-8, Saquinavir 129618-40-2, Nevirapine 132539-06-1, Olanzapine 134678-17-4, Lamivudine 135729-61-2, Palonosetron 136470-78-5, Abacavir 136817-59-9, Delavirdine 137234-62-9, Voriconazole 139110-80-8, Zanamivir 139226-28-1, Darbufelone 139264-17-8, Zolmitriptan 143322-58-1, Eletriptan 143491-57-0, Emtricitabine 144034-80-0, Rizatriptan 147127-20-6, Tenofovir 149908-53-2, Azimilide 150378-17-9, Indinavir 152459-95-5, Imatinib 153559-49-0, Bexarotene 154323-57-6, Almotriptan 154361-50-9, Capecitabine 154598-52-4, Efavirenz 155213-67-5, Ritonavir 158751-64-5, ClAmikalant 159519-65-0, Enfuvirtide 159989-64-7, Nelfinavir 161814-49-9, Amprenavir 162011-90-7, Rofecoxib 169590-42-5, Celecoxib 169944-35-8, Bisulfan 170729-80-3, Aprepitant 181695-72-7, Valdecoxib 184475-35-2, Gefitinib 190017-00-6, Correolide 192725-17-0, Lopinavir 196618-13-0, Oseltamivir 198470-84-7, Parecoxib 198904-31-3, Atazanavir 202409-33-4, Etoricoxib 220991-20-8, Lumiracoxib 226700-79-4, Fosamprenavir 260792-29-8, 6β,7β-Diacetoxy-13-hydroxylabda-8,14-diene 264875-61-8, Cimiracemoside A 501938-01-8, 23-epi-26-Deoxyactein 856012-03-8 865111-98-4 865147-59-7, Altissinone 871260-93-4
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(coated vaginal devices for vaginal delivery of therapeutically effective and/or health-promoting agents)

IT **89987-06-4**, Tiludronate
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(coated vaginal devices for vaginal delivery of therapeutically effective and/or health-promoting agents)

RN 89987-06-4 HCAPLUS

CN Phosphonic acid, [[(4-chlorophenyl)thio]methylene]bis- (9CI) (CA INDEX NAME)



L97 ANSWER 9 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2006:185468 HCAPLUS
DOCUMENT NUMBER: 144:299401
TITLE: Sustained-release anticancer agent for implantation
INVENTOR(S): Kong, Qingzhong
PATENT ASSIGNEE(S): Shandong Lanjin Biotech Co., Ltd., Peop. Rep. China
SOURCE: Faming Zhuanli Shenqing Gongkai Shuomingshu, 10 pp.
CODEN: CNXXEV
DOCUMENT TYPE: Patent
LANGUAGE: Chinese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CN 1660438	A	20050831	CN 2004-10075840	20041229

PRIORITY APPLN. INFO.: CN 2004-10075840 20041229

AB The title anticancer agent contains bischloroethylamines as active components, and biodegradable high polymers as auxiliary agents. This agent can be produced into implant, sustained-release agent, or sustained-release implant. This agent can selectively increase the drug concentration at the tumor site and improve the therapeutic effectiveness of nonoperative therapies including chemotherapy and **radiotherapy.**

IC ICM A61K045-08

CC 63-6 (Pharmaceuticals)
Section cross-reference(s): 1

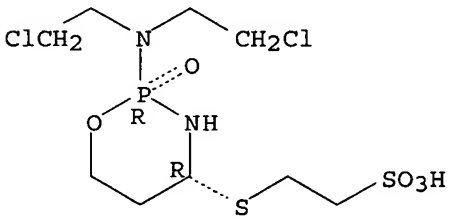
IT 51-18-3, Triamelin 51-79-6, Urethane 54-91-1, Pipobroman 56-25-7, Cantharidin 125-45-1, Azatepa 299-75-2, Treosulfan 302-49-8, Uredepa 602-41-5, Thiocolchicoside 911-45-5, Clomifene 1661-29-6, Meturedepa 1954-28-5, Etoglucid 1980-45-6, Benzodepa 2608-24-4, Piposulfan 3733-81-1, Defosfamide 3778-73-2, Ifosfamide 4148-16-7, Ritrosulfan 4342-03-4, Dacarbazine 5696-17-3, Epipropidine 7518-35-6, Mannosulfan 10087-89-5, Enpromate 13425-98-4, Improsulfan 19039-02-2, Taxodone 22089-22-1, Trofosfamide 29745-04-8, Norcantharidin 36508-71-1, Zorubicin hydrochloride 37753-10-9, Sufosfamide 42061-52-9, Pumitepa 54083-22-6, Zorubicin 62435-42-1, Perfosfamide 69558-55-0, Thymopentin 74550-97-3, Bimolane **88859-04-5**, Mafosfamide 112809-51-5, Letrozole 162011-90-7, Rofecoxib
RL: PAC (Pharmacological activity); PKT (Pharmacokinetics); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(sustained-release anticancer agent for implantation)

IT **88859-04-5**, Mafosfamide
RL: PAC (Pharmacological activity); PKT (Pharmacokinetics); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(sustained-release anticancer agent for implantation)

RN 88859-04-5 HCAPLUS

CN Ethanesulfonic acid, 2-[[(2R,4R)-2-[bis(2-chloroethyl)amino]tetrahydro-2-oxido-2H-1,3,2-oxazaphosphorin-4-yl]thio]-, rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.



L97 ANSWER 10 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2005:667481 HCAPLUS

DOCUMENT NUMBER: 143:333285

TITLE: Particulate assemblies of CdS and TiO2 prepared by Langmuir-Blodgett technique with octadecylamine/methylstearate mixed films

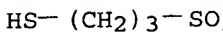
AUTHOR(S): Takahashi, Masashi; Natori, Hirotaka; Tajima, Kazuo; Kobayashi, Koichi

CORPORATE SOURCE: Department of Environmental Energy Engineering, Musashi Institute of Technology, Setagaya, Tokyo, 158-8557, Japan

SOURCE: Thin Solid Films (2005), 489(1-2), 205-214
CODEN: THSFAP; ISSN: 0040-6090

PUBLISHER: Elsevier B.V.

DOCUMENT TYPE: Journal
LANGUAGE: English
AB Langmuir-Blodgett (LB) technique has been applied to fabrications of various nanoparticulate assemblies such as monolayer and multilayer of CdS particles, in-plane mixed monolayer and alternate multilayer of CdS/TiO2 particles on the solid substrate. We examined the most favorable conditions for yielding uniform and dense packing of the particles in a 2-dimensional arrangement, followed by structural characterization of the as-deposited particulate layers. As a result, it was found for the CdS particulate films that charge d. of cationic Langmuir monolayer markedly influences the amount of particles embedded on the substrate and that multilayer LB deposition enables stepwise control of thickness for the particulate assemblies. For the composite CdS/TiO2 particulate films, desired in-plane mixing ratio and alternating layer structure of two kinds of particles were achieved, on which basis the possibilities of fabricating a variety of heteroparticulate layers are pointed out. In addition, photocatalytic activities of TiO2 and CdS/TiO2 particulate films fabricated by the LB technique were evaluated in terms of decomposition of both oleic acid cast film and stearic acid (SA) LB film by **irradiating** with UV light. The results exhibited that when the bilayer of TiO2 particulate film is employed in place of the monolayer film, the photodecompn. rate of SA is sufficiently accelerated and that the alternate CdS/TiO2 particulate layer is less active for the oxidative decomposition
CC 66-3 (Surface Chemistry and Colloids)
IT **49594-30-1D**, 3-Mercapto-1-propanesulfonic acid, cadmium sulfide bound
RL: CPS (Chemical process); PEP (Physical, engineering or chemical process); PROC (Process)
(fabrication of cadmium sulfide and titania nanoparticles by Langmuir-Blodgett method)
IT **49594-30-1D**, 3-Mercapto-1-propanesulfonic acid, cadmium sulfide bound
RL: CPS (Chemical process); PEP (Physical, engineering or chemical process); PROC (Process)
(fabrication of cadmium sulfide and titania nanoparticles by Langmuir-Blodgett method)
RN 49594-30-1 HCAPLUS
CN 1-Propanesulfonic acid, 3-mercapto- (6CI, 9CI) (CA INDEX NAME)



REFERENCE COUNT: 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L97 ANSWER 11 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:1174714 HCAPLUS
DOCUMENT NUMBER: 143:432214
TITLE: In B-CLL, the codon 72 polymorphic variants of p53 are not related to drug resistance and disease prognosis
AUTHOR(S): Sturm, Isrid; Bosanquet, Andrew G.; Hummel, Michael; Doerken, Bernd; Daniel, Peter T.
CORPORATE SOURCE: Department of Hematology, Oncology and Tumor Immunology, University Medical Center Charite, Humboldt University, Berlin, 13353, Germany
SOURCE: BMC Cancer (2005), 5, No pp. given
CODEN: BCMACL; ISSN: 1471-2407
URL: http://www.biomedcentral.com/content/pdf/1471-

2407-5-105.pdf
PUBLISHER: BioMed Central Ltd.
DOCUMENT TYPE: Journal; (online computer file)
LANGUAGE: English

AB Background: A common sequence polymorphism at codon 72 of the p53 gene encoding either arginine or proline was recently shown to be functionally relevant for apoptosis induction in vitro. In B-type chronic lymphocytic leukemia (B-CLL), p53 gene mutations occur in a subset of patients and are associated with impaired survival and drug resistance. Here, we address the functional relevance of the codon 72 single nucleotide (SNP) polymorphism for cell death sensitivity following exposure to clin. employed cytotoxic drugs and γ-irradiation Methods: 138 B-CLL samples were analyzed by SSCP-PCR and sequencing for single nucleotide polymorphism at codon 72 of the p53 gene. The in vitro cytotoxicity assay (DiSC-assay) was performed with 7 drugs (chlorambucil, mafosfamide, fludarabine phosphate, methylprednisolone, doxorubicin, vincristine) or γ-irradiation Results: Of the 138 B-CLL samples, 9 samples were homozygous for proline (Pro/Pro), 78 samples homozygous for arginine (Arg/Arg), and 49 samples heterozygous (Arg/Pro). No differences were found for patient survival and cell death triggered by 7 cytotoxic drugs or γ-irradiation Conclusion: These data indicate that polymorphic variants of p53 codon 72 are not clin. relevant for apoptosis induction or patient survival in B-CLL.

CC 1-6 (Pharmacology)
Section cross-reference(s): 3, 8, 14

ST p53 gene polymorphism antitumor **radiotherapy** resistance leukemia prognosis

IT Codons
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(72 of p53; in B-CLL, codon 72 polymorphic variants of p53 are not related to drug and **radiotherapy** resistance and disease prognosis)

IT Gene, animal
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(TP53; in B-CLL, codon 72 polymorphic variants of p53 are not related to drug and **radiotherapy** resistance and disease prognosis)

IT Drug resistance
(antitumor; in B-CLL, codon 72 polymorphic variants of p53 are not related to drug and **radiotherapy** resistance and disease prognosis)

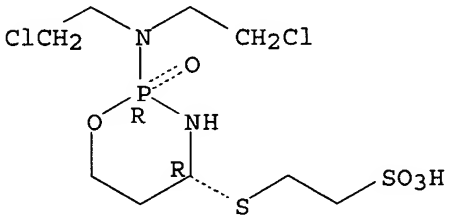
IT **Radiotherapy**
(gamma-ray; in B-CLL, codon 72 polymorphic variants of p53 are not related to drug and **radiotherapy** resistance and disease prognosis)

IT Antitumor agents
Apoptosis
Chronic B-cell leukemia
Cytotoxic agents
Genotypes
Human
Mutation
Prognosis
(in B-CLL, codon 72 polymorphic variants of p53 are not related to drug and **radiotherapy** resistance and disease prognosis)

IT p53 (protein)
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(in B-CLL, codon 72 polymorphic variants of p53 are not related to drug and **radiotherapy** resistance and disease prognosis)

IT Antitumor agents
(resistance to; in B-CLL, codon 72 polymorphic variants of p53 are not related to drug and **radiotherapy** resistance and disease prognosis)
IT Genetic polymorphism
(single nucleotide; in B-CLL, codon 72 polymorphic variants of p53 are not related to drug and **radiotherapy** resistance and disease prognosis)
IT 57-22-7, Vincristine 83-43-2, Methylprednisolone 305-03-3, Chlorambucil 4291-63-8, Cladribine 23214-92-8, Doxorubicin 75607-67-9, Fludarabine phosphate **88859-04-5**, Mafosfamide
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(in B-CLL, codon 72 polymorphic variants of p53 are not related to drug and **radiotherapy** resistance and disease prognosis)
IT **88859-04-5**, Mafosfamide
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(in B-CLL, codon 72 polymorphic variants of p53 are not related to drug and **radiotherapy** resistance and disease prognosis)
RN 88859-04-5 HCAPLUS
CN Ethanesulfonic acid, 2-[[(2R,4R)-2-[bis(2-chloroethyl)amino]tetrahydro-2-oxido-2H-1,3,2-oxazaphosphorin-4-yl]thio]-, rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.



REFERENCE COUNT: 29 THERE ARE 29 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L97 ANSWER 12 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:565039 HCAPLUS
DOCUMENT NUMBER: 141:84767
TITLE: Method for treating patients for **radiation** exposure
INVENTOR(S): Hausheer, Frederick H.
PATENT ASSIGNEE(S): Bionumerik Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 19 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004058103	A1	20040715	WO 2002-US41665	20021220

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,

PL, PT, RO, RU, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA,
UG, US, UZ, VN, YU, ZA, ZM, ZW
RW: AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE,
DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR

CA 2508243	AA	20040715	CA 2002-2508243		20021220
AU 2002360829	A1	20040722	AU 2002-360829		20021220
EP 1581149	A1	20051005	EP 2002-796114		20021220
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK					
CN 1735388	A	20060215	CN 2002-830170		20021220
JP 2006510714	T2	20060330	JP 2004-563165		20021220
PRIORITY APPLN. INFO.:			WO 2002-US41665	W	20021220

OTHER SOURCE(S): MARPAT 141:84767

AB This invention relates to a method of treating a patient suffering from **radiation** exposure, or of prophylactically treating a patient about to undergo **radiation** therapy. The method includes administering to a patient in need of treatment an effective amount of a thiol or reducible disulfide compound according to the formula set forth in the specification.

IC ICM A61F002-02
ICS A61F009-02; A61F013-02; A61K009-20; A61K009-48; A61L015-16

CC 8-9 (Radiation Biochemistry)
Section cross-reference(s): 63

ST **radiotherapy** damage prevention thiol disulfide compd

IT Human
Ionizing **radiation**
Radioprotectants
Radiotherapy
(compds. for treating patients exposed to **radiation**)

IT Disulfides
Thiols, biological studies
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(compds. for treating patients exposed to **radiation**)

IT Drug delivery systems
(injections; compds. for treating patients exposed to **radiation**
)

IT Drug delivery systems
(oral; compds. for treating patients exposed to **radiation**)

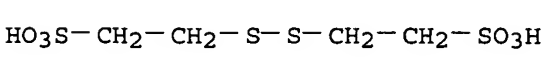
IT Drug delivery systems
(parenterals; compds. for treating patients exposed to **radiation**)

IT 52-90-4D, Cysteine, conjugates 70-18-8D, Glutathione, conjugates
6027-13-0D, Homocysteine, conjugates **16208-51-8**, DiMesna
19767-45-4, Mesna
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(compds. for treating patients exposed to **radiation**)

IT **16208-51-8**, DiMesna
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(compds. for treating patients exposed to **radiation**)

RN 16208-51-8 HCAPLUS

CN Ethanesulfonic acid, 2,2'-dithiobis-, disodium salt (9CI) (CA INDEX NAME)



L97 ANSWER 13 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:452971 HCAPLUS
DOCUMENT NUMBER: 141:22208
TITLE: Prevention and treatment of cancer metastasis and bone loss associated with cancer metastasis
INVENTOR(S): Zimmerman, Deborah Lee; Harrowe, Gregory; Liu, Cheng; Koths, Kirston; Kavanaugh, W. Michael; Long, Li
PATENT ASSIGNEE(S): Chiron Corporation, USA
SOURCE: PCT Int. Appl., 135 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2004045532	A2	20040603	WO 2003-US36679	20031117
WO 2004045532	A3	20060126		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
CA 2505994	AA	20040603	CA 2003-2505994	20031117
AU 2003291002	A1	20040615	AU 2003-291002	20031117
EP 1572106	A2	20050914	EP 2003-783587	20031117
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				

PRIORITY APPLN. INFO.: US 2002-426781P P 20021115
WO 2003-US36679 W 20031117

AB The authors disclose M-CSF antagonists for preventing and treating bone metastases. Also disclosed are methods of screening for M-CSF antagonists and uses of M-CSF in preventing and treating bone metastases and tumor growth. In one example, an antibody to M-CSF ameliorates osteolysis in a mouse model.

IC ICM A61K

CC 15-3 (Immunochemistry)
Section cross-reference(s): 1, 2, 8, 14

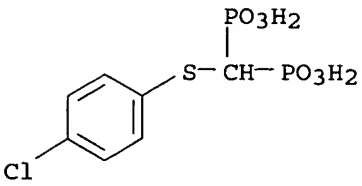
IT **Radiotherapy**
Surgery
(combination therapy with M-CSF antagonists for treatment of cancer metastasis and metastasis-associated bone loss)

IT 2809-21-4 10596-23-3 40391-99-9 66376-36-1, Alendronate **89987-06-4**, Tiludronic acid 114084-78-5, Ibandronate 118072-93-8, Zoledronate 143011-72-7, G-CSF
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(combination therapy with M-CSF antagonists for treatment of cancer metastasis and metastasis-associated bone loss)

IT **89987-06-4**, Tiludronic acid
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(combination therapy with M-CSF antagonists for treatment of cancer metastasis and metastasis-associated bone loss)

RN 89987-06-4 HCAPLUS

CN Phosphonic acid, [[(4-chlorophenyl)thio]methylene]bis- (9CI) (CA INDEX NAME)



L97 ANSWER 14 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:550645 HCAPLUS
DOCUMENT NUMBER: 141:85155
TITLE: Determining the density of functional moieties on polymer reagents
INVENTOR(S): Stetson, Christopher M.; Albarella, James P.; Corey, Paul F.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 7 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004132092	A1	20040708	US 2003-336573	20030103
WO 2004062572	A2	20040729	WO 2003-US39378	20031211
WO 2004062572	A3	20041209		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003296484	A1	20040810	AU 2003-296484	20031211
PRIORITY APPLN. INFO.:			US 2003-336573 A	20030103
			WO 2003-US39378 W	20031211

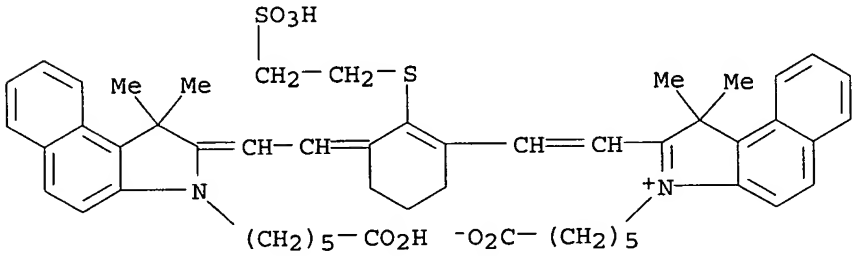
AB The invention provides a method for determining the d. of functional mols. attached to a substrate used for anal. of biol. samples. A dye mol. responding to near IR **radiation** at a wavelength of at least 600 nm is attached to the substrate and used to indicate the number of the functional mols. attached to the substrate by comparing the IR absorption of the dye mols. with the UV absorption of the functional mols. Such substrates may be employed in immunoassays and in vivo diagnostics.
IC ICM G01N033-53
ICS G01N033-574
INCL 435007100; 435007230
CC 9-15 (Biochemical Methods)
IT 9003-01-4, Polyacrylic acid 9004-54-0, Dextrans, analysis 25322-68-3 25702-74-3D, Ficoll, Aminoethylcarbonylmethyl derivs. **345891-45-4**

, DTO 108 716326-43-1 716326-44-2 716326-45-3 716326-46-4
716326-47-5 716326-48-6 716326-49-7 716326-50-0 716326-51-1
716326-52-2 716326-53-3 716326-54-4 716326-55-5 716326-56-6
716326-57-7 716326-58-8 716326-59-9 716326-60-2 716326-61-3
716326-62-4
RL: ARU (Analytical role, unclassified); ANST (Analytical study)
(determining the d. of functional moieties on polymer reagents)

IT **345891-45-4**, DTO 108
RL: ARU (Analytical role, unclassified); ANST (Analytical study)
(determining the d. of functional moieties on polymer reagents)

RN 345891-45-4 HCAPLUS

CN 1H-Benz[e]indolium, 3-(5-carboxypentyl)-2-[2-[3-[[3-(5-carboxypentyl)-1,3-dihydro-1,1-dimethyl-2H-benz[e]indol-2-ylidene]ethylidene]-2-[(2-sulfoethyl)thio]-1-cyclohexen-1-yl]ethenyl]-1,1-dimethyl-, inner salt (9CI) (CA INDEX NAME)



L97 ANSWER 15 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:120569 HCAPLUS
DOCUMENT NUMBER: 140:181315
TITLE: Preparation of furanones as cytoprotectants for dermatologic conditions
INVENTOR(S): Boddupalli, Sekhar; Walkinshaw, Gail; Wang, Bing
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 66 pp., Cont.-in-part of U.S. Ser. No. 354,474.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2004029812	A1	20040212	US 2003-630170	20030730
US 2003176361	A1	20030918	US 2003-354474	20030128
US 6667330	B2	20031223		
WO 2005016340	A1	20050224	WO 2004-US24491	20040728

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE,

SN, TD, TG

EP 1660080 A1 20060531 EP 2004-786136 20040728
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK
PRIORITY APPLN. INFO.: US 2002-353939P P 20020131
US 2003-354474 A2 20030128
US 2003-630170 A 20030730
WO 2004-US24491 W 20040728
OTHER SOURCE(S): MARPAT 140:181315

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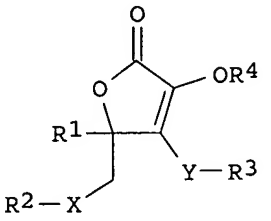

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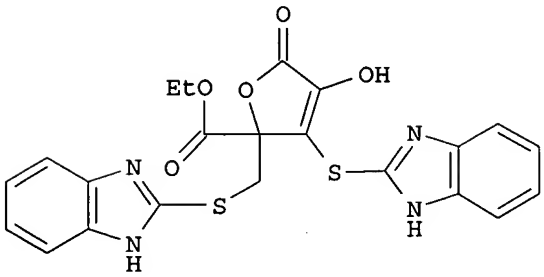


II

AB Title compds. I [R1 = CO2R', CONR'R'', CH2OR''', CN, (un)substituted heterocyclyl, heterocyclylalkyl, heteroaryl, heteroaralkyl; R2, R3 = independently (un)substituted alkyl, cycloalkyl, aryl, aralkyl, heterocyclyl, heteroaryl, heteroaralkyl, nucleoside, amino acid, di-, tri- or tetra-peptide; R4 = H, alkyl, alkylcarbonyl, (poly)alkoxyalkylene, dialkoxyphosphoryloxy; X = alkylene, NR', S, SO, SO2; or XR2 = PO(OR')2; Y = NR', S, SO, SO2; or YR3 = PO(OR')2; or XR2YR3 = (un)substituted aliphatic or aromatic ring; R' = H, alkenyl, (un)substituted alkyl, cycloalkyl, phosphoryl, aryl; R'' = H, alkenyl, (un)substituted alkyl, aryl; or R'R'' = atoms that form (un)substituted 5-7 membered aryl, heteroaryl ring; R''' = H, alkenyl, (un)substituted alkyl, acyl, cycloalkyl, phosphoryl, aryl; and their single tautomers, single stereoisomers, mixts. of tautomers and/or stereoisomers, and pharmaceutically acceptable salts] were prepared as cytoprotectants for treating dermatol. conditions. For example, II was prepared by reaction of 2-mercaptobenzimidazole with Et bromopyruvate in ethanol/acetone and aldol condensation of the two tautomeric forms of the pyruvate intermediate. Selected invention compds. showed significant reduction in edema in assays assessing mouse ear inflammatory response to topical arachidonic acid (10% to 70%, $p < 0.05$). Results from various assays were disclosed for selected invention compds. Thus, I and their pharmaceutical formulations are useful for regulating skin condition, regulating the signs of skin aging or for treating contact dermatitis, skin irritation, acne, rosacea, psoriasis, age-related damage or damage

resulting from harmful (UV) **radiation** or environmental pollution, stress or fatigue.

IC ICM A61K038-06
ICS A61K038-05; A61K038-04; A61K031-541; A61K031-496; A61K031-5377; A61K031-452; A61K031-427; A61K031-421; A61K031-4178; A61K031-4025; A61K031-365

INCL 514018000; 514217030; 514227800; 514231500; 514254100; 514326000; 514365000; 514374000; 514397000; 514422000

CC 27-6 (Heterocyclic Compounds (One Hetero Atom))
Section cross-reference(s): 1, 34, 63

IT 577952-48-8P, 3-(3-Amino-[1,2,4]thiadiazol-5-ylsulfanyl)-2-(((3-amino-[1,2,4]thiadiazol-5-yl)sulfanyl)methyl)-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577952-49-9P, 3-(3-Amino-[1,2,4]thiadiazol-5-ylsulfanyl)-2-(((3-amino-[1,2,4]thiadiazol-5-yl)sulfanyl)methyl)-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester, trimethylamine salt 577952-50-2P, 3-((5-Amino-2H-[1,2,4]triazol-3-yl)sulfanyl)-2-(((5-amino-2H-[1,2,4]triazol-3-yl)sulfanyl)methyl)-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577952-52-4P, 4-Hydroxy-5-oxo-3-(5-phenyl-[1,3,4]oxadiazol-2-ylsulfanyl)-2-(5-phenyl-[1,3,4]oxadiazol-2-ylsulfanylmethyl)-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577952-53-5P, 3-(5-Chlorobenzothiazol-2-ylsulfanyl)-2-[(5-chloro-benzothiazol-2-ylsulfanyl)methyl]-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577952-54-6P, 4-Hydroxy-3-(5-methoxy-1H-benzimidazol-2-ylsulfanyl)-2-[(5-methoxy-1H-benzimidazol-2-ylsulfanyl)methyl]-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577952-55-7P, 4-Hydroxy-5-oxo-3-(p-tolylsulfanyl)-2-(p-tolylsulfanylmethyl)-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577952-56-8P 577952-62-6P 577952-63-7P 577952-64-8P 577952-65-9P 577952-66-0P 577952-67-1P 577952-72-8P 577952-73-9P, 4-Hydroxy-5-oxo-3-(pyridin-4-ylsulfanyl)-2-[(pyridin-4-ylsulfanyl)methyl]-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577952-74-0P, 5,8-Dichloro-3-hydroxy-2-oxo-2H-1-oxa-4,9-dithiabenzo[f]azulene-10a-carboxylic acid ethyl ester 577952-75-1P, 3-(1H-Benzimidazol-2-ylsulfanyl)-2-[(1H-benzimidazol-2-ylsulfanyl)methyl]-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid 577952-76-2P, 3-(Benzothiazol-2-ylsulfanyl)-2-[(benzothiazol-2-ylsulfanyl)methyl]-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid (2-hydroxyethyl)amide 577952-78-4P, 3-(Benzothiazol-2-ylsulfanyl)-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid 577952-79-5P, 4-(Furan-2-ylmethylsulfanyl)-5-[(furan-2-ylmethylsulfanyl)methyl]-3-hydroxy-5-hydroxymethyl-5H-furan-2-one 577952-81-9P, 4-(2,2-Dimethylpropionyloxy)-3-(furan-2-ylmethylsulfanyl)-2-[(furan-2-ylmethylsulfanyl)methyl]-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577952-82-0P 577952-83-1P 577952-85-3P, 4-(1H-Benzimidazol-2-ylsulfanyl)-5-[(1H-benzimidazol-2-ylsulfanyl)methyl]-3-hydroxy-5-(thiazol-2-yl)-5H-furan-2-one 577952-86-4P, 3-(Benzothiazol-2-ylsulfanyl)-2-[(benzothiazol-2-ylsulfanyl)methyl]-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid 577952-87-5P, 3-(2-Chloro-4-fluorophenylsulfanyl)-2-[(2-chloro-4-fluorophenylsulfanyl)methyl]-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577952-88-6P 577952-89-7P, 4-(Benzoxazol-2-ylsulfanyl)-5-[(benzoxazol-2-ylsulfanyl)methyl]-3-hydroxy-5-hydroxymethyl-5H-furan-2-one 577952-90-0P, 4-(5-Chlorobenzothiazol-2-ylsulfanyl)-5-[(5-chlorobenzothiazol-2-ylsulfanyl)methyl]-3-hydroxy-5-hydroxymethyl-5H-furan-2-one 577952-91-1P, 4-(Benzothiazol-2-ylsulfanyl)-5-[(benzothiazol-2-ylsulfanyl)methyl]-3-hydroxy-5-hydroxymethyl-5H-furan-2-one 577952-92-2P, 3-(2-Chloro-6-fluorobenzylsulfanyl)-2-[(2-chloro-6-fluorobenzylsulfanyl)methyl]-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577952-93-3P, 3-(5,6-Dichloro-1H-benzimidazol-2-ylsulfanyl)-2-[(5,6-dichloro-1H-benzimidazol-2-ylsulfanyl)methyl]-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester

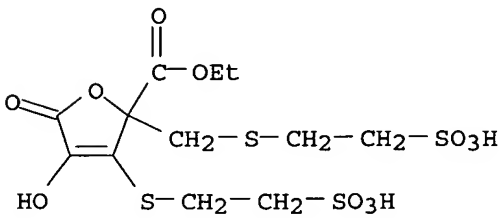
577952-94-4P, 4-Hydroxy-3-(5-methoxybenzothiazol-2-ylsulfanyl)-2-[(5-methoxybenzothiazol-2-ylsulfanyl)methyl]-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577952-95-5P, 3-(2,4-Dichlorobenzylsulfanyl)-2-[(2,4-dichlorobenzylsulfanyl)methyl]-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577952-96-6P, 2-[(Benzothiazol-2-ylsulfinyl)methyl]-3-(benzothiazol-2-ylsulfanyl)-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577952-98-8P, 4-Hydroxy-3-(6-nitrobenzothiazol-2-ylsulfanyl)-2-[(6-nitrobenzothiazol-2-ylsulfanyl)methyl]-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577952-99-9P, 2-[(1H-Benzimidazol-2-ylsulfanyl)methyl]-4-ethoxy-3-(1-ethyl-1H-benzimidazol-2-ylsulfanyl)-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-00-5P, 3-[Furan-2-ylmethanesulfinyl]-2-((furan-2-ylmethanesulfinyl)methyl)-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-01-6P, 2-[(Furan-2-ylmethanesulfinyl)methyl]-3-(furan-2-ylmethanesulfonyl)-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-02-7P, 4-Hydroxy-3-methylsulfanyl-2-methylsulfanylmethyl-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-03-8P, 3-(5-Amino-[1,3,4]thiadiazol-2-ylsulfanyl)-2-(((5-amino-[1,3,4]thiadiazol-2-yl)sulfanyl)methyl)-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid 577953-04-9P, 3-(Benzoxazol-2-ylsulfanyl)-2-[(benzothiazol-2-ylsulfanyl)methyl]-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid methyl ester 577953-05-0P 577953-06-1P 577953-07-2P, 3-(Furan-2-ylmethylsulfanyl)-2-[(furan-2-ylmethylsulfanyl)methyl]-4-isobutanoyloxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-08-3P, 4-(2,2-Dimethylpropanoyloxy)-3-ethoxycarbonylmethylsulfanyl-2-[(ethoxycarbonylmethylsulfanyl)methyl]-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-09-4P, 4-Hydroxy-5-oxo-3-(4-phenylthiazol-2-ylsulfanyl)-2-[(4-phenylthiazol-2-ylsulfanyl)methyl]-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-10-7P, 3-(2-Dimethylaminoethylsulfanyl)-2-[(2-dimethylaminoethylsulfanyl)methyl]-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid 577953-11-8P, 4-Hydroxy-3-[(1-methyl-1H-imidazol-2-yl)sulfanyl]-2-[(1-methyl-1H-imidazol-2-ylsulfanyl)methyl]-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-12-9P, 3-Cyclopentylsulfanyl-2-cyclopentylsulfanylmethyl-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-13-0P, 3-Butylsulfanyl-2-butylsulfanylmethyl-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-14-1P, 4-Hydroxy-3-isobutylsulfanyl-2-isobutylsulfanylmethyl-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-15-2P, 4-Hydroxy-3-(naphthalen-2-ylsulfanyl)-2-[(naphthalen-2-ylsulfanyl)methyl]-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-16-3P, 4-Hydroxy-5-oxo-3-[(1-phenyl-1H-tetrazol-5-yl)sulfanyl]-2-[[(1-phenyl-1H-tetrazol-5-yl)sulfanyl]methyl]-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-17-4P, 4-Hydroxy-5-oxo-3-((5-phenyl-2H-[1,2,4]triazol-3-yl)sulfanyl)-2-(((5-phenyl-2H-[1,2,4]triazol-3-yl)sulfanyl)methyl)-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-18-5P, 4-Hydroxy-5-oxo-3-(thiazol-2-ylsulfanyl)-2-[(thiazol-2-ylsulfanyl)methyl]-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-19-6P, 3-Benzylsulfanyl-2-benzylsulfanylmethyl-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-20-9P, 4-Hydroxy-3-(4-methoxyphenylsulfanyl)-2-[(4-methoxyphenylsulfanyl)methyl]-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-21-0P, 3-(2-Chlorophenylsulfanyl)-2-[(2-chlorophenylsulfanyl)methyl]-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-22-1P, 3-(Benzothiazol-2-ylsulfanyl)-2-[(benzothiazol-2-ylsulfanyl)methyl]-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-23-2P, 3-(Benzoxazol-2-ylsulfanyl)-2-[(benzoxazol-2-ylsulfanyl)methyl]-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-24-3P, 4-Hydroxy-5-oxo-3-(4-trifluoromethylpyrimidin-2-ylsulfanyl)-2-[(4-trifluoromethylpyrimidin-2-ylsulfanyl)methyl]-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-25-4P,

4-Hydroxy-3-(4-methylpyrimidin-2-ylsulfanyl)-2-[(4-methylpyrimidin-2-ylsulfanyl)methyl]-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-26-5P, 4-Hydroxy-5-oxo-3-(pyrimidin-2-ylsulfanyl)-2-[(pyrimidin-2-ylsulfanyl)methyl]-2,5-dihydrofuran-2-carboxylic acid ethyl ester **577953-27-6P**, 4-Hydroxy-5-oxo-3-(2-sulfo-ethylsulfanyl)-2-[(2-sulfo-ethylsulfanyl)methyl]-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-28-7P, 4-Hydroxy-5-oxo-3-(7-trifluoromethylquinolin-4-ylsulfanyl)-2-[(7-trifluoromethylquinolin-4-ylsulfanyl)methyl]-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-29-8P 577953-30-1P 577953-31-2P, 3-Cyclohexylsulfanyl-2-cyclohexylsulfanylmethyl-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-32-3P, 4-(Benzothiazol-2-ylsulfanyl)-5-benzoyl-3-hydroxy-5H-furan-2-one 577953-33-4P, 3-(1H-Benzimidazol-2-ylsulfanyl)-4-hydroxy-5-oxo-5H-furan-2,2-dicarboxylic acid diethyl ester 577953-34-5P, 5-Acetyl-4-(benzothiazol-2-ylsulfanyl)-3-hydroxy-5H-furan-2-one 577953-35-6P, 3-Benzylsulfanyl-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-36-7P, 4-Hydroxy-3-(5-methyl-1H-benzimidazol-2-ylsulfanyl)-5-oxo-2,5-dihydrofuran-2-carboxylic acid 2-isopropyl-5-methylcyclohexyl ester 577953-37-8P 577953-38-9P, 3-(Benzoselenazol-2-ylsulfanyl)-2-[(benzoselenazol-2-ylsulfanyl)methyl]-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-39-0P, 4-Hydroxy-5-oxo-3-(4-phenylthiazol-2-ylsulfanyl)-2,5-dihydrofuran-2-carboxylic acid 577953-40-3P 577953-41-4P, 4-Hydroxy-5-oxo-3-(9H-purin-6-ylsulfanyl)-2-[(9H-purin-6-ylsulfanyl)methyl]-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-42-5P 577953-43-6P, 4-Hydroxy-3-(1H-imidazol-2-ylsulfanyl)-2-[(1H-imidazol-2-ylsulfanyl)methyl]-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-44-7P, 3-(2-Diethylaminoethylsulfanyl)-2-[(2-diethylaminoethylsulfanyl)methyl]-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-45-8P, 3-(1H-Benzimidazol-2-ylsulfanyl)-2-[(1H-benzimidazol-2-ylsulfanyl)methyl]-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid methyl ester 577953-46-9P, 3-(2-Dimethylaminoethylsulfanyl)-2-[(2-dimethylaminoethylsulfanyl)methyl]-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester hydrochloride 577953-47-0P, 4-Hydroxy-3-(2-methoxycarbonylethylsulfanyl)-2-[(2-methoxycarbonylethylsulfanyl)methyl]-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-48-1P, 4-Hydroxy-3-(methoxycarbonylmethylsulfanyl)-2-[(methoxycarbonylmethylsulfanyl)methyl]-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-49-2P, 3-(5-Amino-[1,3,4]thiadiazol-2-ylsulfanyl)-2-[((5-amino-[1,3,4]thiadiazol-2-yl)sulfanyl)methyl]-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-50-5P, 3-(1H-Benzimidazol-2-ylsulfanyl)-2-[(1H-benzimidazol-2-ylsulfanyl)methyl]-4-hydroxy-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-51-6P, 3-(4-Fluorobenzylsulfanyl)-4-hydroxy-5-oxo-2,5-dihydrofuran-2,2-dicarboxylic acid diethyl ester 577953-52-7P, 4-Hydroxy-5-oxo-3-(1-oxopyridin-2-ylsulfanyl)-2-[(1-oxopyridin-2-ylsulfanyl)methyl]-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-53-8P, 4-Hydroxy-3-(4-methoxybenzylsulfanyl)-2-[(4-methoxybenzylsulfanyl)methyl]-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester 577953-54-9P, 4-Hydroxy-3-(5-nitro-1H-benzimidazol-2-ylsulfanyl)-2-((5-nitro-1H-benzimidazol-2-ylsulfanyl)methyl)-5-oxo-2,5-dihydrofuran-2-carboxylic acid ethyl ester
RL: PAC (Pharmacological activity); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(cytoprotective agent; preparation of furanone cytoprotectants via aldol condensation for treatment of dermatol. conditions)

IT **577953-27-6P**, 4-Hydroxy-5-oxo-3-(2-sulfo-ethylsulfanyl)-2-[(2-sulfo-ethylsulfanyl)methyl]-2,5-dihydrofuran-2-carboxylic acid ethyl ester
RL: PAC (Pharmacological activity); SPN (Synthetic preparation); THU

(Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(cytoprotective agent; preparation of furanone cytoprotectants via aldol condensation for treatment of dermatol. conditions)

RN 577953-27-6 HCAPLUS
CN 2-Furancarboxylic acid, 2,5-dihydro-4-hydroxy-5-oxo-3-[(2-sulfoethyl)thio]-2-[[(2-sulfoethyl)thio]methyl]-, 2-ethyl ester (9CI) (CA INDEX NAME)



L97 ANSWER 16 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:690528 HCAPLUS
DOCUMENT NUMBER: 142:106257
TITLE: The treatment of neoplastic meningitis
AUTHOR(S): Armstrong, Terri S.; Gilbert, Mark R.
CORPORATE SOURCE: Department of Neuro-Oncology, The University of Texas MD Anderson Cancer Center, Houston, TX, 77030, USA
SOURCE: Expert Opinion on Pharmacotherapy (2004), 5(9), 1929-1935
CODEN: EOPHF7; ISSN: 1465-6566
PUBLISHER: Ashley Publications Ltd.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review. Neoplastic meningitis (NM) is a debilitating complication of cancer that occurs when tumor cells infiltrate the leptomeninges. Treatment often includes direct installation of chemotherapy into the cerebrospinal fluid either by lumbar puncture or the use of a ventricular reservoir, **radiation** therapy, systemic chemotherapy or a combination of these modalities. The current standard chemotherapeutic agents for direct instillation into the cerebrospinal fluid include methotrexate, cytarabine and thiotepa. Other agents, such as topotecan, manfosfamide and IFNs, are undergoing evaluation in clin. trials. Despite active investigation of new therapies, the prognosis for patients with NM remains poor. However, some patients do demonstrate improvement of neurol. function and prolongation of survival with treatment. Therefore, careful evaluation and treatment planning is warranted in order to avoid treatment-associated toxicities and to maximise the impact of the treatment on the disease process.

CC 1-0 (Pharmacology)
ST review neoplastic meningitis chemotherapy **radiation** therapy anticancer methotrexate
IT Brain
Human
Neoplasm
Radiotherapy
(direct installation of chemotherapy into cerebrospinal fluid by lumbar puncture or use of ventricular reservoir, **radiation** therapy, systemic chemotherapy or combination of these modalities are common treatments for NM in human)
IT Meningitis

(neoplastic; direct installation of chemotherapy into cerebrospinal fluid by lumbar puncture or use of ventricular reservoir, **radiation** therapy, systemic chemotherapy or combination of these modalities are common treatments for NM in human)

IT **88859-04-5**, Mafosfamide
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(direct installation of manfosfamide in to cerebrospinal fluid may be effective for treatment of neoplastic meningitis in human and is under clin. trial)

IT 147-94-4, Cytarabine
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(direct instillation of chemotherapeutic agent cytarabine in to cerebrospinal fluid alone or in combination with **radiation** therapy or systemic chemotherapy are effective treatment for neoplastic meningitis in human)

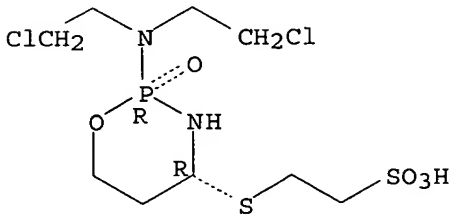
IT 59-05-2, Methotrexate
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(direct instillation of chemotherapeutic agent methotrexate in to cerebrospinal fluid alone or in combination with **radiation** therapy or systemic chemotherapy are effective treatment for neoplastic meningitis in human)

IT 52-24-4, Thiotepa
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(direct instillation of chemotherapeutic agent thiotepa in to cerebrospinal fluid alone or in combination with **radiation** therapy or systemic chemotherapy are effective treatment for neoplastic meningitis in human)

IT **88859-04-5**, Mafosfamide
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(direct installation of manfosfamide in to cerebrospinal fluid may be effective for treatment of neoplastic meningitis in human and is under clin. trial)

RN 88859-04-5 HCAPLUS
CN Ethanesulfonic acid, 2-[[(2R,4R)-2-[bis(2-chloroethyl)amino]tetrahydro-2-oxido-2H-1,3,2-oxazaphosphorin-4-yl]thio]-, rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.



REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L97 ANSWER 17 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:319444 HCAPLUS
DOCUMENT NUMBER: 138:314584
TITLE: Vasostatin fragment from human calreticulin as bone

marrow cell protectant against chemotherapeutic and **radiation** toxicity

INVENTOR(S): Tosato, Giovanna; Pike, Sandra E.; Yao, Lei
PATENT ASSIGNEE(S): The Government of the United States of America, USA
SOURCE: U.S. Pat. Appl. Publ., 47 pp., Cont.-in-part of Appl. No. PCT/US99/23240.
CODEN: USXXCO

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2003078198	A1	20030424	US 2001-828000	20010406
US 6596690	B2	20030722		
WO 2000020577	A1	20000413	WO 1999-US23240	19991005
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, ZA, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2003216299	A1	20031120	US 2003-405588	20030401

PRIORITY APPLN. INFO.:

US 1998-103438P	P	19981006
WO 1999-US23240	A2	19991005
US 2001-828000	A3	20010406

AB The invention is based on the discovery that the N-domain (residues 1-180) of human calreticulin, designated vasostatin, stimulates the proliferation and survival in vitro of hematopoietic cells in the presence of previously identified growth factors. Vasostatin, protects hematopoietic cells in vitro and in vivo from a toxicity induced by chemotherapy or irradiation Bone marrow cell stimulation by vasostatin is observed in the presence of cyclophosphamide (commonly used for treatment of Burkitt lymphoma), maphosphamide (a crosslinker of DNA), methotrexate (an antimetabolite that inhibits dihydrofolic acid reductase), etoposide (an inhibitor of cell cycle progression), and cisplatin (a cell cycle nonspecific interstrand DNA crosslinker). Several active fragments of vasostatin are also disclosed, encompassing amino acids 103-163, amino acids 120-146, and amino acids 129-146. Thus, the invention provides a method of stimulating the proliferation or survival of a hematopoietic cell exposed to a chemotherapeutic agent or irradiation using these fragments.

IC ICM A61K038-17
ICS C12N009-99
INCL 514012000; 435184000
CC 1-8 (Pharmacology)
ST calreticulin fragment bone marrow hematopoietic cell protection; vasostatin bone marrow hematopoietic cell protection; chemotherapy bone marrow hematopoietic cell protection vasostatin; **radiation** bone marrow hematopoietic cell protection vasostatin
IT Crosslinking agents
(DNA; vasostatin fragment from human calreticulin as bone marrow cell protectant against chemotherapeutic and **radiation** toxicity)
IT Hematopoietin receptors
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(FLT3 receptors, co-treatment with; vasostatin fragment from human calreticulin as bone marrow cell protectant against chemotherapeutic

and **radiation** toxicity)

IT Proteins
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(MBP (maltose-binding protein), fusion products; vasostatin fragment from human calreticulin as bone marrow cell protectant against chemotherapeutic and **radiation** toxicity)

IT Calreticulin
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(amino-terminal fragment (vasostatin); vasostatin fragment from human calreticulin as bone marrow cell protectant against chemotherapeutic and **radiation** toxicity)

IT Cytotoxic agents
(antimetabolites; vasostatin fragment from human calreticulin as bone marrow cell protectant against chemotherapeutic and **radiation** toxicity)

IT Growth factors, animal
Interleukin 3
Interleukin 6
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(co-treatment with; vasostatin fragment from human calreticulin as bone marrow cell protectant against chemotherapeutic and **radiation** toxicity)

IT Cell cycle
(inhibitors; vasostatin fragment from human calreticulin as bone marrow cell protectant against chemotherapeutic and **radiation** toxicity)

IT Antibodies and Immunoglobulins
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(monoclonal; vasostatin fragment from human calreticulin as bone marrow cell protectant against chemotherapeutic and **radiation** toxicity)

IT Hematopoietic disorders
(treatment of; vasostatin fragment from human calreticulin as bone marrow cell protectant against chemotherapeutic and **radiation** toxicity)

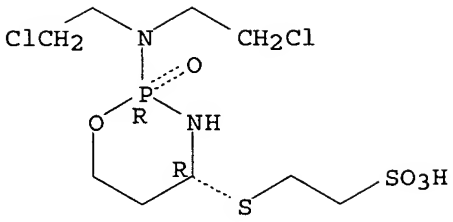
IT Blood cell
Bone marrow
Cell proliferation
Chemotherapy
Drug toxicity
Hematopoietic precursor cell
Hematopoietic precursor cell
Human
Radiotherapy
(vasostatin fragment from human calreticulin as bone marrow cell protectant against chemotherapeutic and **radiation** toxicity)

IT Chemokines
Cytokines
Steroids, biological studies
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(vasostatin fragment from human calreticulin as bone marrow cell protectant against chemotherapeutic and **radiation** toxicity)

IT Calreticulin
Fusion proteins (chimeric proteins)
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(vasostatin fragment from human calreticulin as bone marrow cell protectant against chemotherapeutic and **radiation** toxicity)

IT Interferons
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(α; vasostatin fragment from human calreticulin as bone marrow cell protectant against chemotherapeutic and **radiation** toxicity)
IT 512172-33-7 512172-34-8 512208-07-0, 1-180-Calreticulin (human) 512208-08-1, 103-163-Calreticulin (human) 512208-09-2
RL: PAC (Pharmacological activity); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(amino acid sequence; vasostatin fragment from human calreticulin as bone marrow cell protectant against chemotherapeutic and **radiation** toxicity)
IT 9002-03-3, Dihydrofolate reductase
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(inhibitors; vasostatin fragment from human calreticulin as bone marrow cell protectant against chemotherapeutic and **radiation** toxicity)
IT 512208-30-9
RL: PRP (Properties)
(unclaimed nucleotide sequence; vasostatin fragment from human calreticulin as bone marrow cell protectant against chemotherapeutic and **radiation** toxicity)
IT 512208-31-0 512208-32-1
RL: PRP (Properties)
(unclaimed protein sequence; vasostatin fragment from human calreticulin as bone marrow cell protectant against chemotherapeutic and **radiation** toxicity)
IT 50-18-0, Cyclophosphamide 51-21-8, Fluorouracil 57-22-7, Vincristine 59-05-2, Methotrexate 127-07-1, Hydroxyurea 154-93-8, BiCNU 7440-06-4, Platinum, biological studies 11056-06-7, Bleomycin 15663-27-1, Cisplatinum 21679-14-1, Fludarabine 23214-92-8, Doxorubicin 25316-40-9, Adriamycin 33069-62-4, Taxol 33419-42-0, Etoposide **88859-04-5**, Mafosfamide
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(vasostatin fragment from human calreticulin as bone marrow cell protectant against chemotherapeutic and **radiation** toxicity)
IT 50812-37-8D, Glutathione S-transferase, fusion products with vasostatin 64134-30-1D, Hexahistidine, fusion products with vasostatin
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(vasostatin fragment from human calreticulin as bone marrow cell protectant against chemotherapeutic and **radiation** toxicity)
IT **88859-04-5**, Mafosfamide
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(vasostatin fragment from human calreticulin as bone marrow cell protectant against chemotherapeutic and **radiation** toxicity)
RN 88859-04-5 HCAPLUS
CN Ethanesulfonic acid, 2-[[(2R,4R)-2-[bis(2-chloroethyl)amino]tetrahydro-2-oxido-2H-1,3,2-oxazaphosphorin-4-yl]thio]-, rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.

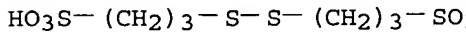


L97 ANSWER 18 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:433139 HCAPLUS
DOCUMENT NUMBER: 138:394020
TITLE: Printed wiring board and its fabrication
INVENTOR(S): Hamada, Tetsuro; Nakamura, Takashi; Kawana, Jun
PATENT ASSIGNEE(S): Toppan Printing Co., Ltd., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 6 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2003163452	A2	20030606	JP 2001-360454	20011127
PRIORITY APPLN. INFO.:			JP 2001-360454	20011127

AB A method for fabricating a printed wiring board having a plated through hole filled with a resin and a lid plating layer on the through hole involves filling a plated through hole of a board with a resin, polishing an excess resin to make the resin planar with the through hole, removing the portion of the resin surface by sand blasting or laser **radiation** to lower the level of the resin surface, roughening the surface of the side wall of the through hole exposed by roughening the resin surface, and forming a lid plating layer by electroless plating and electroplating. Optionally, an electroplating bath containing a strong leveling agent may be used.

IC ICM H05K003-42
ICS C25D003-02; C25D005-16; C25D007-00; H05K001-11
CC 76-2 (Electric Phenomena)
IT 2869-83-2, Janus Green B **17661-52-8**, Bis(3-sulfopropyl)disulfide
RL: NUU (Other use, unclassified); USES (Uses)
(printed wiring board having lid plating layer and its fabrication by electroless plating and electroplating)
IT **17661-52-8**, Bis(3-sulfopropyl)disulfide
RL: NUU (Other use, unclassified); USES (Uses)
(printed wiring board having lid plating layer and its fabrication by electroless plating and electroplating)
RN 17661-52-8 HCAPLUS
CN 1-Propanesulfonic acid, 3,3'-dithiobis- (9CI) (CA INDEX NAME)



L97 ANSWER 19 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:240284 HCAPLUS

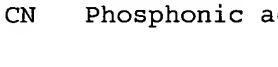
DOCUMENT NUMBER: 138:262472
TITLE: Manufacture of organic electroluminescent device by thermal transferring organic electroluminescent layers via patterned low-thermal-conductive organic compound mask
INVENTOR(S): Yamanaka, Mikihiro
PATENT ASSIGNEE(S): Sharp Corp., Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 10 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 2003092181	A2	20030328	JP 2001-280195	20010914
PRIORITY APPLN. INFO.:			JP 2001-280195	20010914

AB In production of an organic electroluminescent device comprising, successively from the bottom, a substrate, a 1st electrode, organic electroluminescent layers including a light-emitting layer, and a 2nd electrode; the organic electroluminescent layers are formed by thermal transfer process using a mask made of an image-wise patterned organic film with lower thermal conductivity than that of the substrate. In the process, the patterned organic film mask is formed on the substrate having the 1st electrode at first, then organic electroluminescent layers (preferably laminated with a peelable heat-propagating layer) are covered on the top and subjected to heat treatment (e.g., laser scanning) for image-wise thermally transfering the electroluminescent layers, and then the excess electroluminescent layers and the mask pattern are removed. The patterned mask may be made of a macromol. and is formed by lithog. Alternatively, the patterned mask is made of a self-assembled monolayer of a low-mol. weight organic compound and is formed by (1) imagewise **irradiating** the substrate having the 1st electrode with UV to give a superhydrophillic surface, and (2) allowing the low-mol. weight organic compound to adsorptively self assembled on the substrate. The manufacturing process does not include a step of positioning of a metal mask.

IC ICM H05B033-10
ICS G03F007-004; G09F009-00; G09F009-30; H05B033-14; H05B033-22
CC 73-11 (Optical, Electron, and Mass Spectroscopy and Other Related Properties)
Section cross-reference(s): 38, 66, 74
IT UV **radiation**
(for adsorptive self assembly of low-mol. weight organic compound as mask; in manufacture of organic electroluminescent device by thermal transferring organic EL layers via patterned low-thermal-conductive organic compound mask)
IT Laser **radiation**
(heating, for thermally transfer; in manufacture of organic electroluminescent device by thermal transferring organic EL layers via patterned low-thermal-conductive organic compound mask)
IT **135865-74-6** 149918-07-0
RL: REM (Removal or disposal); TEM (Technical or engineered material use); PROC (Process); USES (Uses)
(mask, self-assembled monolayer; in manufacture of organic electroluminescent device by thermal transferring organic EL layers via patterned

low-thermal-conductive organic compound mask)
IT **135865-74-6**
RL: REM (Removal or disposal); TEM (Technical or engineered material use); PROC (Process); USES (Uses)
(mask, self-assembled monolayer; in manufacture of organic electroluminescent device by thermal transferring organic EL layers via patterned low-thermal-conductive organic compound mask)
RN 135865-74-6 HCAPLUS
CN Phosphonic acid, (4-mercaptobutyl)- (9CI) (CA INDEX NAME)

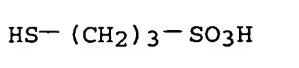


L97 ANSWER 20 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2003:373872 HCAPLUS
DOCUMENT NUMBER: 138:375319
TITLE: Process for electrolytic copper plating
INVENTOR(S): Tsuchida, Hideki; Kusaka, Masaru; Hayashi, Shinjiro; Tsukagoshi, Satoru
PATENT ASSIGNEE(S): Shipley Company LLC, USA
SOURCE: Eur. Pat. Appl., 14 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

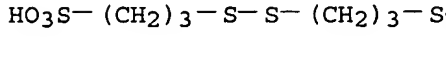
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1310582	A1	20030514	EP 2002-257669	20021106
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, SK				
CN 1432666	A	20030730	CN 2002-154219	20021107
JP 2003213478	A2	20030730	JP 2002-323615	20021107
US 2004089557	A1	20040513	US 2002-289964	20021107
PRIORITY APPLN. INFO.:			JP 2001-341976 A	20011107

AB A process for electrolytic copper plating, that is suitable for the formation of filled vias without compromising the brightness of the deposit is provided. In this process, copper electroplating is carried out in the presence of a transition metal oxide.
IC ICM C25D003-38
ICS H01L021-768
CC 72-8 (Electrochemistry)
Section cross-reference(s): 48, 56
IT UV **radiation**
(electrolytic copper plating without compromising brightness of deposit using)
IT 7664-93-9, Sulfuric acid, uses 16887-00-6, Chloride, uses
17636-10-1, Sodium 3-mercapto-1-propanesulfonate
27206-35-5, Disodium bis(3-sulfopropyl)disulfide
RL: NUU (Other use, unclassified); USES (Uses)
(electrolytic copper plating without compromising brightness of deposit, in solution containing)
IT **17636-10-1**, Sodium 3-mercapto-1-propanesulfonate
27206-35-5, Disodium bis(3-sulfopropyl)disulfide
RL: NUU (Other use, unclassified); USES (Uses)
(electrolytic copper plating without compromising brightness of

deposit, in solution containing)
RN 17636-10-1 HCAPLUS
CN 1-Propanesulfonic acid, 3-mercapto-, monosodium salt (8CI, 9CI) (CA INDEX NAME)



RN 27206-35-5 HCAPLUS
CN 1-Propanesulfonic acid, 3,3'-dithiobis-, disodium salt (9CI) (CA INDEX NAME)



REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L97 ANSWER 21 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:778028 HCAPLUS
DOCUMENT NUMBER: 137:295792
TITLE: A method of treating the surface of a substrate polymer useful for graft polymerization
INVENTOR(S): Kambouris, Peter; Whittaker, Michael; Davis, Tom; Blakey, Idriss; Day, Gary
PATENT ASSIGNEE(S): Polymerat Pty. Ltd., Australia
SOURCE: PCT Int. Appl., 79 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002079305	A1	20021010	WO 2002-AU416	20020328
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2003088028	A1	20030508	US 2002-109777	20020328
US 6858309	B2	20050222		
EP 1383828	A1	20040128	EP 2002-712637	20020328
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,				

IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
PRIORITY APPLN. INFO.: AU 2001-4048 A 20010328
WO 2002-AU416 W 20020328

AB Radicals are generated on functional and/or backbone portions of polymers forming part of a solid phase surface and/or sub-surface to generate a substrate for initiation of polymerization The polymerization is conducted in the presence of a control agent which induces a dynamic population of anchored growing (in a controlled manner) and dormant polymeric chains each comprising ≥2 monomers. Polymers generated by this process include homopolymers and copolymers (comprising ≥2 monomers including terpolymers) such as inter alia block, graft, tapered, crosslinked and branched polymers. The substrate PMA 6100 was **irradiated** from Co-60 source, treated with TEMPO control agent, washed and dried, and graft polymerized with styrene at 80° for 16 h.

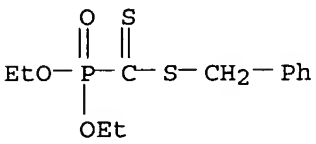
IC ICM C08J007-16
ICS C08J007-18; C08J007-12; C08J009-224; C08J009-36; C08F008-34; C08F008-30; C08F255-02; B29C071-04

CC 38-2 (Plastics Fabrication and Uses)
Section cross-reference(s): 35

IT 366-18-7, 2,2'-Bipyridine 546-68-9, Tetraisopropoxytitanium 558-13-4, Carbon tetrabromide 942-91-6, Carboxymethyl dithiobenzoate 2564-83-2, TEMPO 3030-47-5 3083-10-1 4206-52-4, N-Propyl-2-pyridylmethanimine 5925-55-3, tert-Butyl dithiobenzoate 7032-24-8 12078-28-3, Dicarbonyl(cyclopentadienyl)iodoiron 26504-29-0, Dibenzyl trithiocarbonate 32993-05-8, Chloro(cyclopentadienyl)bis(triphenylphosphine)ruthenium 33527-91-2, Tris[2-(dimethylamino)ethyl]amine 37912-25-7, 1-Phenylethyl dithiobenzoate 72230-93-4, 4,4'-Di(5-nonyl)-2,2'-bipyridine 92361-49-4, Chloro(pentamethylcyclopentadienyl)bis(triphenylphosphine)ruthenium 99897-61-7, Chloro(indenyl)bis(triphenylphosphine)ruthenium 129841-38-9 **178878-93-8** 193557-31-2, N-Pentyl-2-pyridylmethanimine 201611-77-0 201611-79-2 201611-80-5 201611-81-6 201611-82-7 201611-84-9 201611-85-0 201611-90-7 201611-91-8 201611-92-9 377725-60-5 469886-38-2 469886-39-3 469886-41-7 469886-42-8 469886-43-9
RL: CAT (Catalyst use); USES (Uses)
(irradiation of a nonfunctional substrate polymer for graft polymerization with styrene in the presence of one or more control agents)

IT **178878-93-8**
RL: CAT (Catalyst use); USES (Uses)
(irradiation of a nonfunctional substrate polymer for graft polymerization with styrene in the presence of one or more control agents)

RN 178878-93-8 HCAPLUS
CN Phosphinecarbodithioic acid, diethoxy-, phenylmethyl ester, 1-oxide (9CI) (CA INDEX NAME)



REFERENCE COUNT: 13 THERE ARE 13 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L97 ANSWER 22 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:688469 HCAPLUS
DOCUMENT NUMBER: 137:215809
TITLE: Non-myeloablative tolerogenic treatment
INVENTOR(S): Slavin, Shimon; Prigozhina, Tatyana
PATENT ASSIGNEE(S): Hadasit Medical Research Services and Development Ltd., Israel
SOURCE: U.S., 52 pp., Cont.-in-part of U.S. 6,428,782.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6447767	B1	20020910	US 2000-506082	20000216
US 6428782	B1	20020806	US 1998-222011	19981231
PRIORITY APPLN. INFO.:			US 1997-862550	B2 19970523
			US 1998-222011	A2 19981231

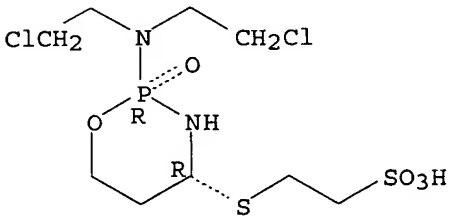
AB The present invention features a method of inducing donor-specific tolerance in a host. Tolerogenic treatments of the present invention may be administered to a host prior to transplantation of donor-derived materials. The tolerogenic treatment involves (1) administering an immunosuppressive agent to a host mammal in a non-myeloablative regimen sufficient to decrease, but not necessarily to eliminate, the host mammal's functional T lymphocyte population; (2) infusing donor antigens from a non-syngeneic donor into the host mammal; (3) eliminating those host T lymphocytes responding to the infused donor antigens using a non-myeloablative dose of lymphocytotoxic or tolerizing agent; and (4) administering donor hematopoietic cells to the host mammal. Donor lymphoid cells used for cell therapy of a host mammal can be depleted of host specific immunol. reactivity by methods essentially similar to those use for tolerizing a host mammal prior to transplantation.

IC ICM A61K038-00
ICS A61K048-00; C12N015-85
INCL 424093100
CC 15-2 (Immunochemistry)
Section cross-reference(s): 1, 14
ST cancer allotransplant tolerance **radiotherapy** lymphocyte
IT Adoptive immunotherapy
Chronic myeloid leukemia
Radiotherapy
(non-myeloablative tolerogenic treatment of cancer and prevention of allograft rejection)
IT **84210-80-0**, ASTA-Z 7557 156586-89-9, Panorex
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(non-myeloablative tolerogenic treatment of cancer and prevention of allograft rejection)
IT **84210-80-0**, ASTA-Z 7557
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(non-myeloablative tolerogenic treatment of cancer and prevention of allograft rejection)
RN 84210-80-0 HCAPLUS
CN Ethanesulfonic acid, 2-[[(2R,4R)-2-[bis(2-chloroethyl)amino]tetrahydro-2-oxido-2H-1,3,2-oxazaphosphorin-4-yl]thio]-, rel-, compd. with cyclohexanamine (1:1) (9CI) (CA INDEX NAME)

CM 1

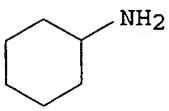
CRN 88859-04-5
CMF C9 H19 Cl2 N2 O5 P S2

Relative stereochemistry.



CM 2

CRN 108-91-8
CMF C6 H13 N



REFERENCE COUNT: 26 THERE ARE 26 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L97 ANSWER 23 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2002:587644 HCAPLUS
DOCUMENT NUMBER: 137:139380
TITLE: Non-myeloablative tolerogenic treatment
INVENTOR(S): Slavin, Shimon; Prigozhina, Tatyana
PATENT ASSIGNEE(S): Hadasit Medical Research Services and Development Ltd., Israel
SOURCE: U.S., 46 pp., Cont.-in-part of U.S. Ser. No. 862,550, abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6428782	B1	20020806	US 1998-222011	19981231
CA 2356434	AA	20000713	CA 1999-2356434	19991223
WO 2000040701	A2	20000713	WO 1999-US30704	19991223
WO 2000040701	A3	20001221		
W: CA, IL, JP, MX				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1141246	A2	20011010	EP 1999-968946	19991223
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

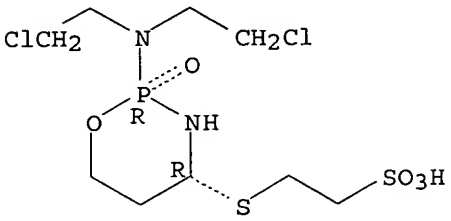
JP 2002534083 T2 20021015 JP 2000-592399 19991223
EP 1498479 A2 20050119 EP 2004-24994 19991223
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY
US 6447767 B1 20020910 US 2000-506082 20000216
EP 1498136 A2 20050119 EP 2004-24995 20041022
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY
PRIORITY APPLN. INFO.: US 1997-862550 B2 19970523
US 1998-222011 A 19981231
EP 1999-968946 A3 19991223
WO 1999-US30704 W 19991223

AB The present invention features a method of inducing donor-specific tolerance in a host. Tolerogenic treatments of the present invention may be administered to a host prior to transplantation of donor-derived materials. The tolerogenic treatment involves (1) administering an immunosuppressive agent to a host mammal in a non-myeloablative regimen sufficient to decrease, but not necessarily to eliminate, the host mammal's functional T lymphocyte population; (2) infusing donor antigens from a non-syngeneic donor into the host mammal; (3) eliminating those host T lymphocytes responding to the infused donor antigens using a non-myeloablative dose of lymphocytotoxic or tolerizing agent; and (4) administering donor hematopoietic cells to the host mammal. Donor lymphoid cells used for cell therapy of a host mammal can be depleted of host specific immunol. reactivity by methods essentially similar to those use for tolerizing a host mammal prior to transplantation.

IC ICM A61K038-00
ICS C12N005-08
INCL 424093100
CC 15-8 (Immunochemistry)
Section cross-reference(s): 1, 8
IT Adoptive immunotherapy
Antitumor agents
Bone marrow
Chronic myeloid leukemia
Hematopoietic precursor cell
Hodgkin's disease
Human
Immune tolerance
Immunosuppressants
Immunosuppression
Immunotherapy
Lymphocyte
Mammalia
Radiotherapy
T cell (lymphocyte)
(procedure for the non-myeloablative tolerogenic prevention of allograft or xenograft rejection)
IT 50-18-0, Cyclophosphamide **88859-04-5**, Mafosfamide
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(procedure for the non-myeloablative tolerogenic prevention of allograft or xenograft rejection)
IT **88859-04-5**, Mafosfamide
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(procedure for the non-myeloablative tolerogenic prevention of allograft or xenograft rejection)
RN 88859-04-5 HCAPLUS
CN Ethanesulfonic acid, 2-[[(2R,4R)-2-[bis(2-chloroethyl)amino]tetrahydro-2-

oxido-2H-1,3,2-oxazaphosphorin-4-yl]thio]-, rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.



REFERENCE COUNT: 66 THERE ARE 66 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L97 ANSWER 24 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:300514 HCAPLUS
DOCUMENT NUMBER: 134:331617
TITLE: Oil-in-water emulsion compositions for polyfunctional active ingredients
INVENTOR(S): Chen, Feng-jing; Patel, Mahesh V.
PATENT ASSIGNEE(S): Lipocine, Inc., USA
SOURCE: PCT Int. Appl., 82 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001028555	A1	20010426	WO 2000-US28835	20001018
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 2002107265	A1	20020808	US 1999-420159	19991018
US 6720001	B2	20040413		

PRIORITY APPLN. INFO.: US 1999-420159 A 19991018

AB Pharmaceutical oil-in-water emulsions for delivery of polyfunctional active ingredients with improved loading capacity, enhanced stability, and reduced irritation and local toxicity are described. Emulsions include an aqueous phase, an oil phase comprising a structured triglyceride, and an emulsifier. The structured triglyceride of the oil phase is substantially free of triglycerides having three medium chain (C6-C12) fatty acid moieties, or a combination of a long chain triglyceride and a polarity-enhancing polarity modifier. The present invention also provides methods of treating an animal with a polyfunctional active ingredient, using dosage forms of the pharmaceutical emulsions. For example, an emulsion was prepared, with cyclosporin A as the polyfunctional active ingredient dissolved in an oil phase including a structured triglyceride (Captex 810D) and a long chain triglyceride (safflower oil). The composition contained (by weight) cyclosporin A 1.0, Captex 810D 5.0, safflower oil 5.0,

BHT 0.02, egg phospholipid 2.4, dimyristoylphosphatidyl glycerol 0.2, glycerol 2.25, EDTA 0.01, and water up to 100%, resp.

IC ICM A61K031-355
ICS A61K031-20

CC 63-6 (Pharmaceuticals)

IT Antibacterial agents
Beverages
Buffers
Chelating agents
Coloring materials
Emulsifying agents
Encapsulation
Evaporation
Extrusion, nonbiological
Filtration
Flavoring materials
Freeze drying
Homogenization
Melting
Mixing
Odor and Odorous substances
Partition
Preservatives
Radiation
Size reduction
Solubilization
Solubilizers
Solvents
Sonication
Spraying
Sterilization and Disinfection
Vaccines
(oil-in-water emulsion compns. for polyfunctional active ingredients)

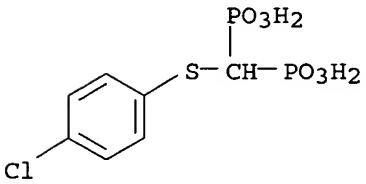
IT 59865-13-3, Cyclosporin A 60142-96-3, Gabapentin 61270-78-8, Cefonicid sodium 61361-72-6, Dimyristoylphosphatidyl glycerol 61379-65-5, Rifapentine 61489-71-2, Menotropin 61869-08-7, Paroxetine 62013-04-1, Dirithromycin 62356-64-3 62893-19-0, Cefoperazone 63527-52-6, Cefotaxime 63585-09-1, Foscarnet sodium 63590-64-7, Terazosin 63612-50-0, Nilutamide 63675-72-9, Nisoldipine 64228-81-5, Atracurium besylate 64544-07-6, Cefuroxime axetil 65271-80-9, Mitoxantrone 65277-42-1, Ketoconazole 66376-36-1, Alendronate 66419-50-9, Bovine growth hormone 68099-86-5, Bepridil hydrochloride 68401-81-0, Ceftizoxime 68506-86-5, Vigabatrin 69049-74-7, Nedocromil sodium 69655-05-6, Didanosine 69756-53-2, Halofantrine 70288-86-7, Ivermectin 70458-92-3, Pefloxacin 70458-96-7, Norfloxacin 71486-22-1, Vinorelbine 72432-03-2, Miglitol 72559-06-9, Rifabutine 73384-59-5, Ceftriaxone 73590-58-6, Omeprazole 73963-72-1, Cilostazol 74011-58-8, Enoxacin 74103-06-3, Ketorolac 74356-00-6, Cefotetan disodium 74381-53-6, Leuprolide acetate 75706-12-6, Leflunomide 76420-72-9, Enalaprilat 76470-66-1, Loracarbef 76547-98-3, Lisinopril 76824-35-6, Famotidine 76963-41-2, Nizatidine 78110-38-0, Aztreonam 79350-37-1, Cefixime 79517-01-4, Octreotide acetate 79617-96-2, Sertraline 79794-75-5, Loratadine 79902-63-9, Simvastatin 81093-37-0, Pravastatin 81098-60-4, Cisapride 81103-11-9, Clarithromycin 81161-17-3, Esmolol hydrochloride 82410-32-0, Ganciclovir 82419-36-1, Ofloxacin 82626-48-0, Zolpidem 82952-64-5, Trimetrexate glucuronate 83799-24-0, Fexofenadine 83869-56-1, Granulocyte-macrophage colony stimulating factor 83881-51-0, Cetirizine 83905-01-5, Azithromycin 84057-84-1, Lamotrigine 84371-65-3, Mifepristone 84449-90-1, Raloxifene 84625-61-6, Itraconazole

85721-33-1, Ciprofloxacin 86386-73-4, Fluconazole 86541-75-5, Benazepril 87679-37-6, Trandolapril 88669-04-9, Trospectomycin 89778-26-7, Toremifene **89987-06-4**, Tiludronate 90357-06-5, Bicalutamide 91161-71-6, Terbinafine 93390-81-9, Fosphenytoin 93413-69-5, Venlafaxine 93479-97-1, Glimepiride 93957-54-1, Fluvastatin 94749-08-3, Salmeterol xinafoate 95233-18-4, Atovaquone 97240-79-4, Topiramate 97322-87-7, Troglitazone 97682-44-5, Irinotecan 98079-51-7, Lomefloxacin 98319-26-7, Finasteride 100986-85-4, Levofloxacin 101828-21-1, Butenafine 103577-45-3, Lansoprazole 103628-46-2, Sumatriptan 104227-87-4, Famciclovir 104987-11-3, Tacrolimus 105462-24-6, Risedronic acid 106133-20-4, Tamsulosin 106650-56-0, Sibutramine 106819-53-8, Doxacurium chloride 106861-44-3, Mivacurium chloride 107648-80-6, Cefepime hydrochloride 107753-78-6, Zafirlukast 110871-86-8, Sparfloxacin 111025-46-8, Pioglitazone 111406-87-2, Zileuton 112965-21-6, Calcipotriene 113189-02-9, Antihemophilic factor 113665-84-2, Clopidogrel 113852-37-2, Cidofovir 115103-54-3, Tiagabine 116094-23-6, Insulin aspart 117976-89-3, Rabeprazole 118072-93-8, Zoledronate 118292-40-3, Tazarotene 119914-60-2, Grepafloxacin 120014-06-4, Donepezil 121368-58-9, Olpadronate 121679-13-8, Naratriptan 122320-73-4, Rosiglitazone 123948-87-8, Topotecan 124832-26-4, Valaciclovir 127759-89-1, Lobucavir 127779-20-8, Saquinavir 129497-78-5, Verteporfin 131918-61-1, Paricalcitol 133040-01-4, Eprosartan 133107-64-9, Insulin lispro 134523-00-5, Atorvastatin 134678-17-4, Lamivudine 135062-02-1, Repaglinide 137862-53-4, Valsartan 138402-11-6, Irbesartan 139110-80-8, Zanamivir 139264-17-8, Zolmitriptan 139481-59-7, Candesartan 139639-23-9, Tissue type plasminogen activator 143003-46-7, Alglucerase 143011-72-7, Granulocyte colony stimulating factor 144034-80-0, Rizatriptan 144494-65-5, Tirofiban 144701-48-4, Telmisartan 145599-86-6, Cerivastatin 145941-26-0, Oprelvekin 146961-76-4, Alatrofloxacin 147059-72-1, Trovafloxacin 148553-50-8, Pregabalin 151126-32-8, Pramlintide 153559-49-0, Targretin 154361-50-9, Capecitabine 154598-52-4, Efavirenz 155213-67-5, Ritonavir 156259-68-6, Capmul MCM 157810-81-6, Indinavir sulfate 158747-02-5, Frovatriptan 158966-92-8, Montelukast 159989-64-7, Nelfinavir 160337-95-1, Insulin glargine 162011-90-7, Rofecoxib 165101-51-9, Becaplermin 169148-63-4, Insulin detemir 169590-42-5, Celecoxib 173146-27-5, Denileukin diftitox 191588-94-0, TNK-tPA 208666-87-9, Captex 810D
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(oil-in-water emulsion compns. for polyfunctional active ingredients)

IT **89987-06-4**, Tiludronate
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(oil-in-water emulsion compns. for polyfunctional active ingredients)

RN 89987-06-4 HCAPLUS

CN Phosphonic acid, [[(4-chlorophenyl)thio]methylene]bis- (9CI) (CA INDEX NAME)



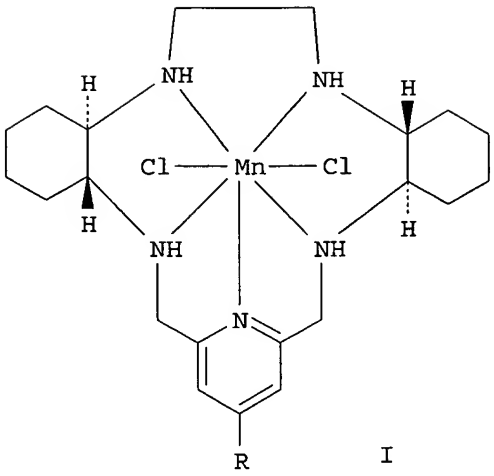
REFERENCE COUNT: 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L97 ANSWER 25 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:257991 HCAPLUS
DOCUMENT NUMBER: 134:274987
TITLE: Substituted pyridino pentaazamacrocycle complexes having superoxide dismutase activity as therapeutic agents
INVENTOR(S): Riley, Dennis P.; Neumann, William L.; Henke, Susan L.; Lennon, Patrick; Aston, Karl W.; Salvemini, Daniela; Sikorski, James A.; Fobian, Yvette M.; Grapperhaus, Margaret Lanahan; Kusturin, Carrie L.
PATENT ASSIGNEE(S): Monsanto Company, USA
SOURCE: U.S., 51 pp., Cont.-in-part of U.S. Ser. No. 57,831.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 5
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 6214817	B1	20010410	US 1999-398120	19990916
US 6180620	B1	20010130	US 1998-57831	19980409
CA 2382105	AA	20010322	CA 2000-2382105	20000914
WO 2001019823	A2	20010322	WO 2000-US25154	20000914
WO 2001019823	A3	20010907		
WO 2001019823	C2	20020926		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1212323	A2	20020612	EP 2000-966722	20000914
EP 1212323	B1	20040609		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
JP 2003509423	T2	20030311	JP 2001-523400	20000914
EP 1420022	A1	20040519	EP 2004-3746	20000914
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
EP 1420019	A1	20040519	EP 2004-3751	20000914
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI, CY				
AT 268774	E	20040615	AT 2000-966722	20000914
PT 1212323	T	20041029	PT 2000-966722	20000914
ES 2222925	T3	20050216	ES 2000-966722	20000914
AU 784078	B2	20060202	AU 2000-77024	20000914
HK 1046689	A1	20050311	HK 2002-108379	20021119

PRIORITY APPLN. INFO.:		
US 1997-50402P	P	19970620
US 1998-57831	A2	19980409
US 1999-398120	A	19990916
EP 2000-966722	A3	20000914
WO 2000-US25154	W	20000914

OTHER SOURCE(S): MARPAT 134:274987
GI



I

AB The present invention relates to compds. which are effective as catalysts for dismutating superoxide and, more particularly, the Mn or Fe complexes of substituted, unsatd. heterocyclic pentaazacyclopentadecane ligands which catalytically dismutate superoxide. The present invention is directed to low mol. weight catalysts, e.g., I (R = cyclohexyl, StBu, SCH2CH2NH2, etc.), for the dismutation of superoxide radicals (SOD mimics) useful as therapeutic agents for inflammatory disease states and disorders in which superoxide anions are implicated. The SOD mimics are Mn or Fe complexes of N-containing 15-membered macrocycle ligands which comprise a substituted, unsatd., N-containing heterocyclic moiety, most preferably those with cyclohexyl, hydroxyl, alkylthio, alkyl 2-thioacetate, benzyloxy, methoxyarylthio, alkoxycarbonylarylthio, and aryl 2-thioacetate substituents. Preferably, the N-containing heterocyclic moiety is aromatic, more preferably, a pyridino moiety. Novel methods of modifying the substituents on the heterocyclic moiety after chelation with the metal ion are also presented. Addition of substituents to the unsatd. N-containing heterocyclic moiety on the pentaazacyclopentadecane macrocycle in the above complexes can drastically alter both the superoxide dismutase catalytic activity and increase the efficacy of these complexes as pharmaceutical agents. The compds. of the invention exhibit a marked increase in potency for the prevention or reversal of opioid tolerance as compared to previously disclosed complexes with unsubstituted N-containing heterocyclic moieties. These compds. are <10 times more potent as pharmaceutical agents for antiinflammatory and analgesic compns. and are as good as, or often better than, the parent unsubstituted compds. in applications such as treatment of endotoxin-induced refractory hypotension. Specific diseases or disorders for which the compds. are claimed as pharmaceutical agents include reperfusion injury to the ischemic myocardium, general inflammation, inflammatory bowel disease, rheumatoid arthritis, osteoarthritis, hypertension, psoriasis, organ transplant rejection, organ preservation, **radiation**-induced injury, platelet aggregation, stroke, autoimmune diseases, carcinogenesis, severe chronic pain, reversal of opioid tolerance, hyperalgesia, and sepsis. Two exemplary formulations for topical application are presented.

IC ICM C07D487-22
ICS A61K031-675; A61K047-16

INCL 514186000

CC 78-7 (Inorganic Chemicals and Reactions)

Section cross-reference(s): 1, 7, 28, 63, 67

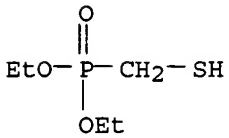
IT Injury
(**radiation**-induced; preparation of manganese substituted pyridino pentaazacyclopentadecane complexes as SOD mimics for treatment of)

IT 60-23-1, 2-Mercaptoethylamine 74-88-4, Methyl iodide, reactions 75-03-6, Ethyl iodide 75-33-2, 2-Mercaptopropane 100-38-9, 2-Diethylaminoethanethiol 107-22-2, Glyoxal 110-89-4, Piperidine, reactions 138-60-3, Chelidamic acid 513-53-1, 2-Mercaptobutane 623-51-8, Ethyl thioglycolate 762-04-9, Diethyl phosphite 931-51-1, Cyclohexylmagnesium chloride 1569-69-3, Cyclohexylmercaptan 2043-61-0, Cyclohexanecarboxaldehyde 2365-48-2, Methyl thioglycolate 4521-31-7, 2-Mercaptobenzyl alcohol 6956-50-9, Ethyl 4,4-dimethoxy-3-oxobutyrate 7217-59-6, 2-Methoxythiophenol 7773-01-5, Manganese dichloride 15570-12-4, 3-Methoxythiophenol 19721-22-3, 3-Mercapto-1-propanol 20439-47-8, (1R,2R)-Diaminocyclohexane 20938-74-3, N-Methylmercaptoacetamide 28276-32-6, Ethyl 4-mercaptobenzoate 41651-93-8, Ethyl 3-mercaptobenzoate **70660-05-8**, Diethyl mercaptomethylphosphonate 330626-95-4 331718-73-1
RL: RCT (Reactant); RACT (Reactant or reagent)
(for preparation of manganese substituted pyridino pentaazacyclopentadecane complexes)

IT 218791-27-6P 301664-32-4P 311767-57-4P 330626-39-6P 330626-40-9P 330626-47-6P **330626-49-8P** 330626-51-2P 330626-54-5P 330626-55-6P 330626-56-7P 330626-58-9P 330626-60-3P 330626-61-4P 330626-62-5P 330626-63-6P 330626-64-7P 330626-65-8P 330626-67-0P 330626-68-1P 330626-71-6P 330626-72-7P 330626-74-9P 330626-75-0P 330626-76-1P 331718-72-0P
RL: CAT (Catalyst use); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of manganese/iron substituted pyridino pentaazacyclopentadecane complexes as SOD mimics for treatment of superoxide-related diseases or disorders)

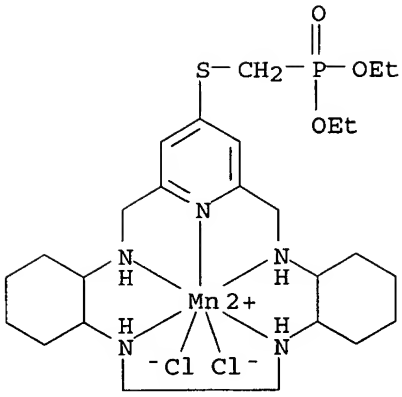
IT **70660-05-8**, Diethyl mercaptomethylphosphonate
RL: RCT (Reactant); RACT (Reactant or reagent)
(for preparation of manganese substituted pyridino pentaazacyclopentadecane complexes)

RN 70660-05-8 HCAPLUS
CN Phosphonic acid, (mercaptomethyl)-, diethyl ester (9CI) (CA INDEX NAME)



IT **330626-49-8P**
RL: CAT (Catalyst use); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of manganese/iron substituted pyridino pentaazacyclopentadecane complexes as SOD mimics for treatment of superoxide-related diseases or disorders)

RN 330626-49-8 HCAPLUS
CN Manganese, dichloro[diethyl [[[(4aR,13aR,17aR,21aR)-1,2,3,4,4a,5,6,12,13,13a,14,15,16,17,17a,18,19,20,21,21a-eicosahydro-11,7-nitrilo-7H-dibenzo[b,h][1,4,7,10]tetraazacycloheptadecin-9-yl-κN5,κN13,κN18,κN21,κN22]thio]methyl]phosphonate]-, (PB-7-11-2344'3')- (9CI) (CA INDEX NAME)



REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L97 ANSWER 26 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:472078 HCAPLUS
DOCUMENT NUMBER: 135:58137
TITLE: Test strip for the assay of an analyte in a liquid sample
INVENTOR(S): Corey, Paul F.; Pugia, Michael J.; Rehm, Gary E.
PATENT ASSIGNEE(S): Bayer Corp., USA
SOURCE: Eur. Pat. Appl., 23 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

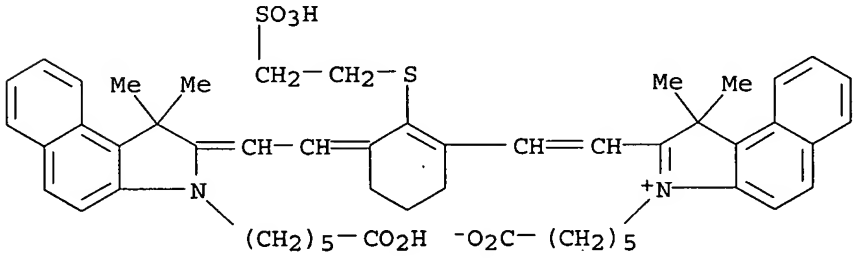
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1111386	A2	20010627	EP 2000-126414	20001205
EP 1111386	A3	20021218		
EP 1111386	B1	20050309		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
US 6316264	B1	20011113	US 1999-466637	19991217
CA 2327127	AA	20010617	CA 2000-2327127	20001130
AT 290693	E	20050315	AT 2000-126414	20001205
ES 2238244	T3	20050901	ES 2000-126414	20001205
AU 778320	B2	20041125	AU 2000-72123	20001208
JP 2001194368	A2	20010719	JP 2000-381173	20001215

PRIORITY APPLN. INFO.: US 1999-466637 A 19991217

AB An improved test strip for determining the presence or concentration of unknown or a constituent in a liquid test sample is disclosed. The test strip comprises a support strip and a test pad, wherein the test pad includes a carrier matrix incorporating a reagent composition capable of interacting with the constituent of interest to produce a detectable or measurable response. The test strip further comprises an IR dye, applied either to the support strip or incorporated into a test pad, which ensures proper alignment of the test strip in an apparatus having a detection system for the detectable or measurable response. The improved test strip reduces the number of erroneous assays for the constituent of interest.

IC ICM G01N033-558

ICS G01N033-52; G01N033-72; G01N033-84
CC 9-1 (Biochemical Methods)
IT Blood analysis
Cameras
Carriers
Color
Composition
Concentration (condition)
IR **radiation**
Illumination
Optical scanners
Spectrometers
(test strip for assay of analyte in a liquid sample)
IT 53655-17-7, 5,5'-Dichloro-11-diphenylamino-3,3'-diethyl-10,12-ethylenethiatricarbocyanine perchlorate 105528-25-4 155773-67-4 **345891-45-4** 345891-46-5
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(test strip for assay of analyte in a liquid sample)
IT **345891-45-4**
RL: ARG (Analytical reagent use); ANST (Analytical study); USES (Uses)
(test strip for assay of analyte in a liquid sample)
RN 345891-45-4 HCAPLUS
CN 1H-Benz[e]indolium, 3-(5-carboxypentyl)-2-[2-[3-[[3-(5-carboxypentyl)-1,3-dihydro-1,1-dimethyl-2H-benz[e]indol-2-ylidene]ethylidene]-2-[(2-sulfoethyl)thio]-1-cyclohexen-1-yl]ethenyl]-1,1-dimethyl-, inner salt (9CI) (CA INDEX NAME)



L97 ANSWER 27 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2001:765004 HCAPLUS
DOCUMENT NUMBER: 136:134835
TITLE: Synthesis of new phosphonyl/S-methyl ketene thioacetals and N-substituted phosphonyl/S-methyl thiocarbonates under microwave **irradiation**
AUTHOR(S): Chen, Kai; Yang, Hua-Zheng; Liu, Zhun; Hu, Fang-Zhong; Zhang, Chun-Xiang
CORPORATE SOURCE: State Key Lab. of Elemento-Organic Chemistry, Inst. of Elemento-Organic Chem., Nankai Univ., Tianjin, 300071, Peop. Rep. China
SOURCE: Youji Huaxue (2001), 21(9), 690-692
CODEN: YCHHDX; ISSN: 0253-2786
PUBLISHER: Kexue Chubanshe
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
OTHER SOURCE(S): CASREACT 136:134835
AB Phosphonyl/S-Me ketene thioacetals, XYC:C(SMe){P(O)(OR)2} (X = CN, Y = CN, CO2Et, PhCO, R = Et; X = Y = CN, R = i-Pr), and N-substituted/S-Me thiocarbonates, X(NC)C:C(SMe){P(O)(OR)2} (X = cyano, 2-ClC6H4CO, R = Et; X

= cyano, R = i-Pr), novel kinds of synthons for the synthesis of phosphonyl heterocyclic compds., were conveniently synthesized under microwave irradiation with high yields. It showed that the reaction process was significantly enhanced using microwave heating.

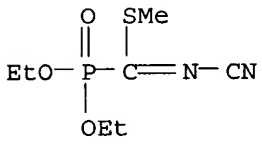
CC 29-7 (Organometallic and Organometalloidal Compounds)

IT 194095-91-5P 194095-94-8P **291545-64-7P** 393110-22-0P
393110-23-1P **393110-24-2P 393110-25-3P**
RL: SPN (Synthetic preparation); PREP (Preparation)
(preparation of)

IT **291545-64-7P 393110-24-2P 393110-25-3P**
RL: SPN (Synthetic preparation); PREP (Preparation)
(preparation of)

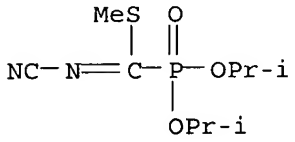
RN 291545-64-7 HCAPLUS

CN Phosphinecarboximidothioic acid, N-cyano-1,1-diethoxy-, methyl ester, 1-oxide (9CI) (CA INDEX NAME)



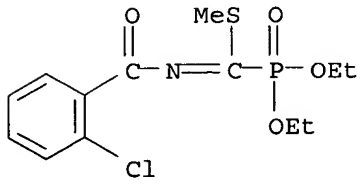
RN 393110-24-2 HCAPLUS

CN Phosphinecarboximidothioic acid, N-cyano-1,1-bis(1-methylethoxy)-, methyl ester, 1-oxide (9CI) (CA INDEX NAME)



RN 393110-25-3 HCAPLUS

CN Phosphinecarboximidothioic acid, N-(2-chlorobenzoyl)-1,1-diethoxy-, methyl ester, 1-oxide (9CI) (CA INDEX NAME)



L97 ANSWER 28 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2000:493415 HCAPLUS

DOCUMENT NUMBER: 133:101470

TITLE: Compositions and methods for the treatment of metabolic bone disorders and bone metastases

INVENTOR(S): Chen, James

PATENT ASSIGNEE(S): Light Sciences, Ltd., USA

SOURCE: PCT Int. Appl., 27 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

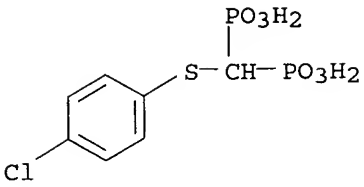
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000041725	A2	20000720	WO 2000-US848	20000114
WO 2000041725	A3	20001130		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
CA 2358662	AA	20000720	CA 2000-2358662	20000114
EP 1131100	A2	20010912	EP 2000-903278	20000114
EP 1131100	B1	20030312		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2002534483	T2	20021015	JP 2000-593335	20000114
AT 234114	E	20030315	AT 2000-903278	20000114
PRIORITY APPLN. INFO.:			US 1999-116233P P	19990115
			WO 2000-US848 W	20000114

OTHER SOURCE(S): MARPAT 133:101470

AB The present invention is drawn to methods and compns. useful for targeting and treating target tissues affected by or involved in metabolic bone disorders and bone metastases with photodynamic therapy (PDT) in a mammalian subject. The compns. are bisphosphonates, pyrophosphates or bisphosphonate-like compds. conjugated to photosensitive agents which are optionally further conjugated to ligands which are target tissue specific antibodies, peptides or polymers. The methods of PDT treatment utilize these compns. to target the tissues or cells of a mammalian subject to be treated. The methods comprise **irradiating** at least a portion of the subject with light at a wavelength absorbed by said photosensitizing agent that under conditions of activation during photodynamic therapy using a relatively low fluence rate, but an overall high total fluence dose results in minimal collateral tissue damage.

IC ICM A61K041-00
CC 8-9 (Radiation Biochemistry)
Section cross-reference(s): 63
IT 61-73-4, Methylene blue 66-97-7D, Psoralen, derivs. 92-31-9, Toluidine blue 106-60-5, δ-Aminolevulinic acid 553-12-8, Protoporphyrin 574-93-6D, Phthalocyanine, derivs. 2683-84-3D, Chlorin, derivs. 2809-21-4 3599-32-4, Indocyanine green 10596-23-3 40391-99-9 66376-36-1, Alendronate 75775-33-6D, Purpurin, derivs. 87806-31-3, Porfimer sodium **89987-06-4**, Tiludronate 105462-24-6 114084-78-5, Ibandronate 129497-78-5, BPD-MA
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(compns. and methods for treatment of metabolic bone disorders and bone metastases)
IT **89987-06-4**, Tiludronate
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(compns. and methods for treatment of metabolic bone disorders and bone metastases)
RN 89987-06-4 HCAPLUS
CN Phosphonic acid, [[(4-chlorophenyl)thio]methylene]bis- (9CI) (CA INDEX

NAME)



L97 ANSWER 29 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:475773 HCAPLUS
DOCUMENT NUMBER: 133:84267
TITLE: Non-myeloablative tolerogenic treatment
INVENTOR(S): Slavin, Shimon; Prigozhina, Tatyana
PATENT ASSIGNEE(S): Hadasit Medical Research Services and Development Ltd., Israel; Baxter International Inc.
SOURCE: PCT Int. Appl., 119 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 4
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000040701	A2	20000713	WO 1999-US30704	19991223
WO 2000040701	A3	20001221		
W: CA, IL, JP, MX				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
US 6428782	B1	20020806	US 1998-222011	19981231
CA 2356434	AA	20000713	CA 1999-2356434	19991223
EP 1141246	A2	20011010	EP 1999-968946	19991223
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2002534083	T2	20021015	JP 2000-592399	19991223

PRIORITY APPLN. INFO.:

US 1998-222011	A	19981231
US 1997-862550	B2	19970523
WO 1999-US30704	W	19991223

AB The present invention features a method of inducing donor-specific tolerance in a host. Tolerogenic treatments of the present invention may be administered to a host prior to transplantation of donor-derived materials. The tolerogenic treatment involves (1) administering an immunosuppressive agent to a host mammal in a non-myeloablative regimen sufficient to decrease, but not necessarily to eliminate, the host mammal's functional T lymphocyte population; (2) infusing donor antigens from a non-syngeneic donor into the host mammal; (3) eliminating those host T lymphocytes responding to the infused donor antigens using a non-myeloablative dose of lymphocytotoxic or inducing tolerance agent; and (4) administering donor hematopoietic cells to the host mammal. Donor lymphoid cells used for cell therapy of a host mammal can be depleted of host specific immunol. reactivity by methods essentially similar to those used for inducing tolerance a host mammal prior to transplantation.

IC ICM C12N005-08
ICS A61K035-12; A61K035-28; A61K039-00; A61P037-02
CC 1-7 (Pharmacology)

Section cross-reference(s): 15

IT **Radiotherapy**
(x-ray, T lymphocyte depletion with, in immune tolerance induction; non-myeloablative induction of tolerance using donor antigens and immunosuppressants to prevent transplantation rejection)

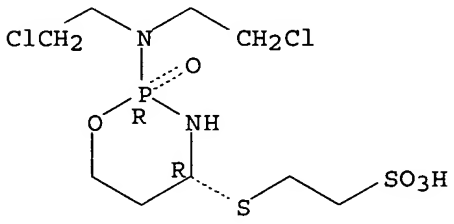
IT 50-18-0, Cyclophosphamide 21679-14-1, Fludarabine **88859-04-5**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(non-myeloablative induction of tolerance using donor antigens and immunosuppressants to prevent transplantation rejection)

IT **88859-04-5**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(non-myeloablative induction of tolerance using donor antigens and immunosuppressants to prevent transplantation rejection)

RN 88859-04-5 HCAPLUS

CN Ethanesulfonic acid, 2-[[(2R,4R)-2-[bis(2-chloroethyl)amino]tetrahydro-2-oxido-2H-1,3,2-oxazaphosphorin-4-yl]thio]-, rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.



L97 ANSWER 30 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN

ACCESSION NUMBER: 2000:807752 HCAPLUS

DOCUMENT NUMBER: 133:329576

TITLE: Method for detection of the effect of different chemotherapeutic agents and/or **radiotherapy** for malignant illnesses, and method for the selection of effective therapy

INVENTOR(S): Daniel, Peter; Hillebrand, Timo; Dorken, Bernd; Bendzko, Peter

PATENT ASSIGNEE(S): Theragen Molekularmedizinische Informationssysteme A.-G., Germany

SOURCE: Ger. Offen., 10 pp.
CODEN: GWXXBX

DOCUMENT TYPE: Patent

LANGUAGE: German

FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 19922052	A1	20001116	DE 1999-19922052	19990514
WO 2000070085	A2	20001123	WO 2000-DE1444	20000510
WO 2000070085	A3	20010809		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV,

MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG,
SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW,
AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE,
DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF,
CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

EP 1181394 A2 20020227 EP 2000-941910 20000510
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO

PRIORITY APPLN. INFO.: DE 1999-19922052 A 19990514
WO 2000-DE1444 W 20000510

AB A method is provided for the detection of the effect of different chemotherapeutic agents and/or **radiotherapy** for malignant illnesses, whereby the expression profiles for tumor- and/or cell growth- and/or apoptosis-associated genes and/or individual differences (mutations) in the gene sequences are determined. Changes in connection with chemotherapeutic agents and/or **radiotherapy** are identified, represented and evaluated diagnostically. Also provided is a method for the selection of effective therapeutic means for the therapy of malignant illnesses. The status of cell cycle genes and/or by apoptosis-associated target genes or their gene products in body fluids, cells, and/or organs is determined and diagnostically evaluated in regard to their effect on suitable therapeutic means. In a preferred embodiment, Bax and p53 expression and/or mutations are examined and therefrom derived are recommendations for individual-specific therapy decisions for leukemia and other malignant illnesses.

IC ICM C12Q001-68
ICS A61K048-00

CC 1-6 (Pharmacology)
Section cross-reference(s): 8

ST chemotherapy **radiotherapy** monitoring malignant disease; antitumor therapy screening gene expression profile; apoptosis gene antitumor therapy monitoring; cell growth gene antitumor therapy monitoring; tumor gene antitumor therapy monitoring; cell cycle gene antitumor therapy monitoring; Bax p53 gene antitumor therapy monitoring

IT Gene, animal
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(ATM; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Promoter (genetic element)
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(Bax, and Bax regulators; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Proteins, specific or class
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(Bax, and gene; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Proteins, specific or class
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(IAP (inhibitor of apoptosis protein), and gene; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Antitumor agents

Chemotherapy
Drug screening
(Method for detection of the effect of different chemotherapeutic agents and/or **radiotherapy** for malignant illnesses, and method for the selection of effective therapy)

IT Gene, animal
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(RB1; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Transcription factors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(Rb; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Gene, animal
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(TP53; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Cyclins
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(and genes; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Neoplasm
(and precancers; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Nutrients
(anti-; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Gene, animal
Proteins, specific or class
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(bcl-2; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Antitumor agents
Antitumor agents
(brain; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Cell proliferation
(cell growth-regulating gene; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Alkylating agents, biological
Animal cell
Body fluid
Cytotoxic agents
Mutation
Organ, animal
Radiotherapy

(chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Anthracyclines
Taxanes
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT DNA
Gene, animal
RNA
p53 (protein)
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Antitumor agents
(chronic lymphocytic leukemia; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT DNA
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(damage, DNA damaging agents; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Antitumor agents
(digestive tract; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Gene
(expression; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Antitumor agents
(female reproductive tract; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Reproductive tract
Reproductive tract
(female, neoplasm, inhibitors; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Apoptosis
(gene regulating; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Cell cycle
(genes; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Antitumor agents
(hematol.; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Steroids, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)

(hormones; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Brain, neoplasm
Brain, neoplasm
Lung, neoplasm
Lung, neoplasm
Pancreas, neoplasm
Pancreas, neoplasm
Skin, neoplasm
Skin, neoplasm
(inhibitors; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Antitumor agents
(leukemia; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Antitumor agents
Antitumor agents
(lung; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Digestive tract
Digestive tract
Endocrine system
Prostate gland
Prostate gland
(neoplasm, inhibitors; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Cyclin dependent kinase inhibitors
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(p16INK4, and gene; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Antitumor agents
Antitumor agents
(pancreas; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Proliferation inhibition
(proliferation inhibitors; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Antitumor agents
(prostate gland; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Antitumor agents
(sarcoma; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Antitumor agents
Antitumor agents
(skin; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Antitumor agents

(solid tumor; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Hormones, animal, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(steroid; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT Alkaloids, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(vinca; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT 150428-23-2, Cyclin-dependent kinase 186322-81-6, Caspase
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(and gene; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

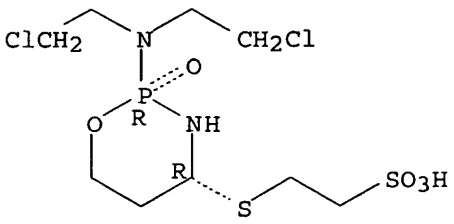
IT 50-24-8, Prednisolone 57-22-7, Vincristine 83-43-2, Methylprednisolone 148-82-3, Melphalan 305-03-3, Chlorambucil 4291-63-8, Cladribine 7440-06-4D, Platinum, compds., biological studies 15663-27-1, Cisplatin 21679-14-1, Fludarabine 23214-92-8, Doxorubicin 56420-45-2, Epirubicin 75607-67-9, Fludarabine phosphate **88859-04-5**, Mafosfamide
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT 80449-01-0, Topoisomerase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

IT **88859-04-5**, Mafosfamide
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(chemotherapeutic agent and/or **radiotherapy** effect detection for malignant illness, and method for selection of effective therapy)

RN 88859-04-5 HCAPLUS
CN Ethanesulfonic acid, 2-[[(2R,4R)-2-[bis(2-chloroethyl)amino]tetrahydro-2-oxido-2H-1,3,2-oxazaphosphorin-4-yl]thio]-, rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.



L97 ANSWER 31 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:115117 HCAPLUS
DOCUMENT NUMBER: 132:273979
TITLE: Ras-Related GTPase RhoB Forces Alkylation-Induced Apoptotic Cell Death
AUTHOR(S): Fritz, Gerhard; Kaina, Bernd
CORPORATE SOURCE: Division of Applied Toxicology, Institute of Toxicology, University of Mainz, Mainz, D-55131, Germany
SOURCE: Biochemical and Biophysical Research Communications (2000), 268(3), 784-789
CODEN: BBRCA9; ISSN: 0006-291X
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB RhoB encoding a Ras-related GTPase is immediate-early inducible by genotoxic treatments. To address the question of the physiol. role of RhoB in cellular defense, cells stably overexpressing wild-type RhoB protein were generated. Overexpression of RhoB renders cells hypersensitive to the killing effect of alkylating agents including antineoplastic drugs but not to UV-light and doxorubicin. As compared to control cells, RhoB overexpressing cells revealed an increase in the frequency of alkylation-induced apoptotic cell death. This indicates that RhoB is involved in modulating apoptotic signaling. Furthermore, overexpression of RhoB resulted in a prolonged transient block to DNA replication upon MMS treatment. UV-induced replication blockage was not affected by RhoB. Based on the data we suggest RhoB to be a novel regulatory factor which takes influence on the level of cytotoxicity of DNA damaging drugs and forces cells to alkylation-induced apoptosis. The data indicate that this might be due to RhoB mediated delay in cell cycle progression upon alkylation treatment. (c) 2000 Academic Press.

CC 1-6 (Pharmacology)
Section cross-reference(s): 13

ST RhoB cytoprotection alkylating antitumor agent apoptosis; methyl methanesulfonate mafosfamide methylnitronitrosoguanidine genotoxicity RhoB drug resistance; cisplatin treosulfan hydrogen peroxide **radiation** DNA damage RhoB

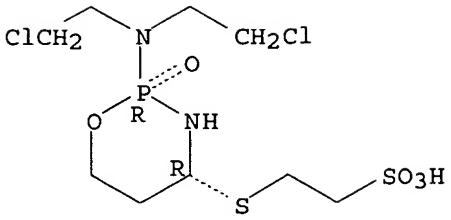
IT Genotoxicity
Ionizing **radiation**
(RhoB in cellular response to genotoxic agent-induced DNA damage)

IT 66-27-3, Methyl methanesulfonate 70-25-7, N-Methyl-N'-nitro-N-nitrosoguanidine 299-75-2, Treosulfan 15663-27-1, Cisplatin **88859-04-5**, Mafosfamide
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(RhoB in cellular response to genotoxic agent-induced DNA damage)

IT **88859-04-5**, Mafosfamide
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(RhoB in cellular response to genotoxic agent-induced DNA damage)

RN 88859-04-5 HCAPLUS
CN Ethanesulfonic acid, 2-[[(2R,4R)-2-[bis(2-chloroethyl)amino]tetrahydro-2-oxido-2H-1,3,2-oxazaphosphorin-4-yl]thio]-, rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.



REFERENCE COUNT: 43 THERE ARE 43 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L97 ANSWER 32 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1999:42478 HCAPLUS
DOCUMENT NUMBER: 130:92218
TITLE: Hydroxymethyl phosphine compounds, and preparation thereof, for use as diagnostic and therapeutic pharmaceuticals
INVENTOR(S): Katti, Kattesh V.; Karra, Srinivasa Rao; Berning, Douglas E.; Smith, C. Jeffrey; Volkert, Wynn A.; Ketring, Alan R.
PATENT ASSIGNEE(S): The Curators of the University of Missouri, USA
SOURCE: U.S., 34 pp., Cont.-in-part of U.S. Ser. No. 412,470, abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

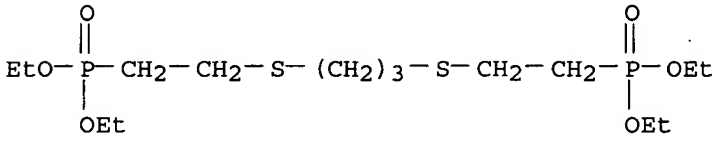
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5855867	A	19990105	US 1997-818080	19970314
CA 2215833	AA	19961003	CA 1996-2215833	19960307
US 5876693	A	19990302	US 1997-902829	19970730
US 6054115	A	20000425	US 1998-33928	19980303
CA 2277179	AA	19980924	CA 1998-2277179	19980305
WO 9841242	A1	19980924	WO 1998-US4318	19980305
W: AU, CA, JP				
RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
AU 9865429	A1	19981012	AU 1998-65429	19980305
EP 1009447	A1	20000621	EP 1998-911487	19980305
EP 1009447	B1	20050810		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2001516360	T2	20010925	JP 1998-540558	19980305
AT 301477	E	20050815	AT 1998-911487	19980305

PRIORITY APPLN. INFO.:
US 1995-412470 B2 19950329
US 1997-818080 A3 19970314
US 1997-902829 A1 19970730
WO 1998-US4318 W 19980305

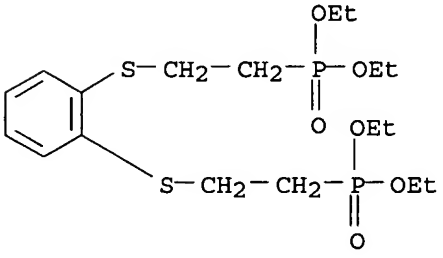
OTHER SOURCE(S): MARPAT 130:92218

AB A compound, and method of making a compound, for use as a diagnostic or therapeutic pharmaceutical comprises at least one functionalized hydroxyalkyl phosphine donor group and one or more sulfur or nitrogen donor and a metal combined with the ligand. Preparation and characterization of ligands and e.g. 99mTc complexes are described. The compds. are useful for therapeutic and diagnostic radiopharmaceuticals.

IC ICM A61K051-00
ICS C07F009-02; C07F005-00; C07C233-00
INCL 424001770
CC 8-9 (Radiation Biochemistry)
Section cross-reference(s): 29, 63, 78
IT Crystal structure
Immobilization, biochemical
Pharmacokinetics
Radiopharmaceuticals
Radiotherapy
Scintigraphic agents
Stability
(hydroxymethyl phosphine compds., and preparation thereof, for use as diagnostic and therapeutic pharmaceuticals)
IT **188107-60-0P 188107-61-1P** 188107-62-2P,
4,8-Dithia-1,11-diphosphaundecane **193073-56-2P** 193073-60-8P,
4,9-Dithia-1,12-diphosphadodecane 193073-65-3P **193073-68-6P**
193073-73-3P, 5,9-Dithia-1,13-diphosphatridecane 193073-77-7P
213772-25-9P 213772-30-6P 213772-37-3P 213772-45-3P 219552-59-7P
219552-67-7P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation and reaction; hydroxymethyl phosphine compds., and preparation thereof, for use as diagnostic and therapeutic pharmaceuticals)
IT **188107-60-0P 188107-61-1P 193073-56-2P**
193073-68-6P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation and reaction; hydroxymethyl phosphine compds., and preparation thereof, for use as diagnostic and therapeutic pharmaceuticals)
RN 188107-60-0 HCAPLUS
CN Phosphonic acid, [1,3-propanediylbis(thio-2,1-ethanediyl)]bis-, tetraethyl ester (9CI) (CA INDEX NAME)

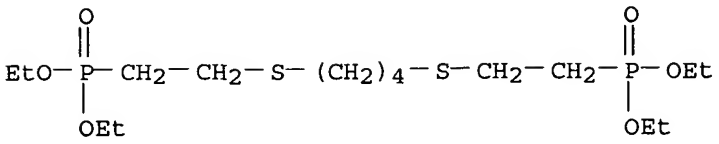


RN 188107-61-1 HCAPLUS
CN Phosphonic acid, [1,2-phenylenebis(thio-2,1-ethanediyl)]bis-, tetraethyl ester (9CI) (CA INDEX NAME)

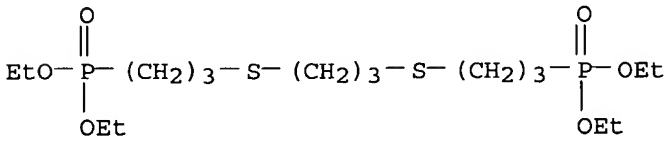


RN 193073-56-2 HCAPLUS
CN Phosphonic acid, [1,4-butanediylbis(thio-2,1-ethanediyl)]bis-, tetraethyl

ester (9CI) (CA INDEX NAME)



RN 193073-68-6 HCAPLUS
CN Phosphonic acid, [1,3-propanediylbis(thio-3,1-propanediyl)]bis-, tetraethyl ester (9CI) (CA INDEX NAME)



REFERENCE COUNT: 36 THERE ARE 36 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L97 ANSWER 33 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2000:14413 HCAPLUS
DOCUMENT NUMBER: 132:44646
TITLE: Total-body **irradiation** and melphalan is a safe and effective conditioning regimen for autologous bone marrow transplantation in children with acute myeloid leukemia in first remission
AUTHOR(S): Bonetti, F.; Zecca, M.; Pession, A.; Messina, C.; Montagna, D.; Lanino, E.; Fagioli, F.; Santoro, N.; Prete, A.; Cesaro, S.; Rondelli, R.; Giorgiani, G.; De Stefano, P.; Locatelli, F.
CORPORATE SOURCE: Italian Association for Pediatric Hematology and Oncology-Bone Marrow Transplantation Group, Department of Pediatrics, University of Pavia, IRCCS Policlinico San Matteo, Pavia, I-27100, Italy
SOURCE: Journal of Clinical Oncology (1999), 17(12), 3729-3735
CODEN: JCONDN; ISSN: 0732-183X
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal
LANGUAGE: English

AB To evaluate the safety and efficacy of a preparative regimen consisting of fractionated total-body **radiation** (9.9 to 12 Gy) and melphalan (140 mg/m2 in a single dose) in children with acute myeloid leukemia in first complete remission (CR) given autologous bone marrow transplantation (ABMT). Fifty-three children (30 males and 23 females; age range, 1.5 to 18 yr) were enrolled onto the study. The median time from first CR to ABMT was 3.5 mo (range, 1.4 to 13 mo), with 45 patients (85%) undergoing transplantation within 6 mo from the diagnosis. Forty-five patients received in vitro marrow purging with standard-dose mafos-famide (100 μg/mL), seven patients were treated with interleukin-2 before marrow collection, and in the remaining child, the marrow was unmanipulated. The median infused cell dose was 1.8 + 108/kg (range, 0.4 to 5.8 + 108/kg). All patients but one achieved hematopoietic engraftment, with a median time to neutrophil recovery of 24 days (range, 11 to 66 days). Treatment-related toxicity was moderate and consisted mainly of mucositis.

One patient died from cytomegalovirus interstitial pneumonia, and one died from pulmonary hemorrhage. Fourteen patients (26%) relapsed at a median time of 6 mo after ABMT (range, 2 to 17 mo), with a cumulative relapse probability of 29% (95% confidence interval, 16% to 42%). The 5-yr Kaplan-Meier estimate of survival for all 53 patients was 78% (range, 65% to 90%), whereas the overall 5-yr disease-free survival was 68% (range, 55% to 81%), with a median follow-up duration of 40 mo (range, 7 to 130 mo). These data suggest that, in our cohort of patients, the combination of total-body irradiation and melphalan is safe and associated with good antileukemia activity, making ABMT an appealing alternative for postremission therapy in children with acute myeloid leukemia in first CR.

CC 1-6 (Pharmacology)
Section cross-reference(s): 8

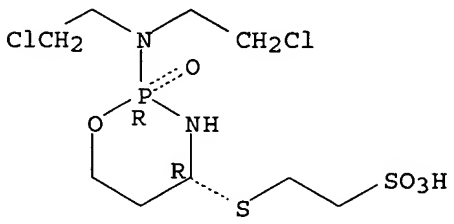
IT Drug tolerance
Radiotherapy
(effect of total-body irradiation and melphalan for autologous bone marrow transplantation in children with acute myeloid leukemia in first remission)

IT 51-48-9, L-Thyroxin, biological studies 148-82-3, Melphalan
88859-04-5, Mafos-famide
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(effect of total-body irradiation and melphalan for autologous bone marrow transplantation in children with acute myeloid leukemia in first remission)

IT **88859-04-5**, Mafos-famide
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(effect of total-body irradiation and melphalan for autologous bone marrow transplantation in children with acute myeloid leukemia in first remission)

RN 88859-04-5 HCAPLUS
CN Ethanesulfonic acid, 2-[[(2R,4R)-2-[bis(2-chloroethyl)amino]tetrahydro-2-oxido-2H-1,3,2-oxazaphosphorin-4-yl]thio]-, rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.

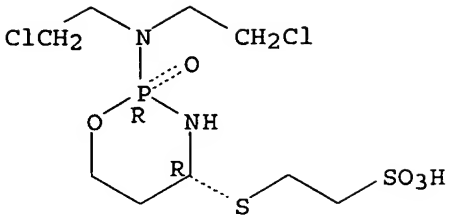


REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L97 ANSWER 34 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1999:153954 HCAPLUS
DOCUMENT NUMBER: 130:308474
TITLE: Activation of c-Jun N-terminal kinase 1 by UV **irradiation** is inhibited by wortmannin without affecting c-jun expression
AUTHOR(S): Fritz, G.; Kaina, B.
CORPORATE SOURCE: Institute of Toxicology, Division of Applied

Toxicology, University of Mainz, Mainz, D-55131, Germany
SOURCE: Molecular and Cellular Biology (1999), 19(3), 1768-1774
CODEN: MCEBD4; ISSN: 0270-7306
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Activation of c-Jun N-terminal kinases (JNKs)/stress-activated protein kinases is an early response of cells upon exposure to DNA-damaging agents. JNK-mediated phosphorylation of c-Jun is currently understood to stimulate the transactivating potency of AP-1 (e.g., c-Jun/c-Fos; c-Jun/ATF-2), thereby increasing the expression of AP-1 target genes. Here we show that stimulation of JNK1 activity is not a general early response of cells exposed to genotoxic agents. Treatment of NIH 3T3 cells with UV light (UV-C) as well as with Me methanesulfonate (MMS) caused activation of JNK1 and an increase in c-Jun protein and AP-1 binding activity, whereas antineoplastic drugs such as mafosfamide, mitomycin C, N-hydroxyethyl-N-chloroethylnitrosourea, and treosulfan did not elicit this response. The phosphatidylinositol 3-kinase inhibitor wortmannin specifically blocked the UV-stimulated activation of JNK1 but did not affect UV-driven activation of extracellular regulated kinase 2 (ERK2). To investigate the significance of JNK1 for transactivation of c-jun, we analyzed the effect of UV irradiation on c-jun expression under conditions of wortmannin-mediated inhibition of UV-induced stimulation of JNK1. Neither the UV-induced increase in c-jun mRNA, c-Jun protein, and AP-1 binding nor the activation of the collagenase and c-jun promoters was affected by wortmannin. In contrast, the mitogen-activated protein kinase/ERK kinase inhibitor PD98059, which blocked ERK2 but not JNK1 activation by UV irradiation, impaired UV-driven c-Jun protein induction and AP-1 binding. Based on the data, we suggest that JNK1 stimulation is not essential for transactivation of c-jun after UV exposure, whereas activation of ERK2 is required for UV-induced signaling leading to elevated c-jun expression.
CC 8-6 (Radiation Biochemistry)
Section cross-reference(s): 1, 4
ST UV **radiation** wortmannin JNK1 ERK2 cjun
IT Mutagens
UV C **radiation**
(activation of c-Jun N-terminal kinase 1 by UV irradiation is inhibited by wortmannin without affecting c-jun expression)
IT 50-07-7, Mitomycin C 299-75-2, Treosulfan **88859-04-5**, Mafosfamide 128202-04-0
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(stimulation of JNK1 activity is not a general early response of cells exposed to genotoxic agents)
IT **88859-04-5**, Mafosfamide
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(stimulation of JNK1 activity is not a general early response of cells exposed to genotoxic agents)
RN 88859-04-5 HCAPLUS
CN Ethanesulfonic acid, 2-[[(2R,4R)-2-[bis(2-chloroethyl)amino]tetrahydro-2-oxido-2H-1,3,2-oxazaphosphorin-4-yl]thio]-, rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.



REFERENCE COUNT: 52 THERE ARE 52 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L97 ANSWER 35 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1999:349876 HCAPLUS
DOCUMENT NUMBER: 131:141486
TITLE: The sulfhydryl containing compounds WR-2721 and glutathione as radio- and chemoprotective agents. A review, indications for use and prospects
AUTHOR(S): Hospers, G. A. P.; Eisenhauer, E. A.; De Vries, E. G. E.
CORPORATE SOURCE: Division of Medical Oncology, Department of Internal Medicine, University Hospital Groningen, Groningen, 9700 RB, Neth.
SOURCE: British Journal of Cancer (1999), 80(5/6), 629-638
CODEN: BJCAAI; ISSN: 0007-0920
PUBLISHER: Churchill Livingstone
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review with over 80 refs. Radio- and chemotherapy for the treatment of malignancies are often associated with significant toxicity. One approach to reduce the toxicity is the concomitant treatment with chemoprotective agents. This article reviews two sulfhydryl compds., namely the agent WR-2721 (amifostine), a compound recently registered for use in human in many countries, and the natural occurring compound glutathione (GSH). GSH is not registered as a chemoprotective agent. WR-2721 is an aminothiol prodrug and has to be converted to the active compound WR-1065 by membrane-bound alkaline phosphatase. WR-1065 and GSH both act as naturally occurring thiols. No protective effect on the tumor has been found when these compds. are administered i.v. There is even in vitro evidence for an increased anti-tumor effect with mafosfamide after pretreatment with WR-2721, and in vivo after treatment with carboplatin and paclitaxel. Randomized clin. studies have shown that WR-2721 and GSH decrease cisplatin-induced nephrotoxicity and that WR-2721 reduces **radiation radiotherapy**-induced toxicity. Side-effects associated with WR-2721 are nausea, vomiting and hypotension, GSH has no side-effects. An exact role of WR-2721 and GSH as chemoprotectors is not yet completely clear. Future studies should examine the protective effect of these drugs on mucositis, cardiac toxicity, neuro- and ototoxicity, the development of secondary neoplasms and their effect on quality of life.

CC 8-0 (Radiation Biochemistry)
ST review sulfhydryl compd **radioprotective** chemoprotective antitumor
IT Cytoprotective agents
Drug interactions
Radioprotectants
Radiotherapy
(sulfhydryl containing compds. as radio- and chemoprotective agents, and potentiating antitumor drug effects)

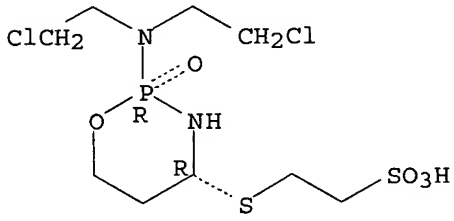
IT 70-18-8, Glutathione, biological studies 20537-88-6, WR-2721
31098-42-7, WR-1065 33069-62-4, Paclitaxel 41575-94-4, Carboplatin
88859-04-5, Mafosfamide
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(sulfhydryl containing compds. as radio- and chemoprotective agents, and potentiating antitumor drug effects)

IT **88859-04-5**, Mafosfamide
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(sulfhydryl containing compds. as radio- and chemoprotective agents, and potentiating antitumor drug effects)

RN 88859-04-5 HCAPLUS

CN Ethanesulfonic acid, 2-[[(2R,4R)-2-[bis(2-chloroethyl)amino]tetrahydro-2-oxido-2H-1,3,2-oxazaphosphorin-4-yl]thio]-, rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.

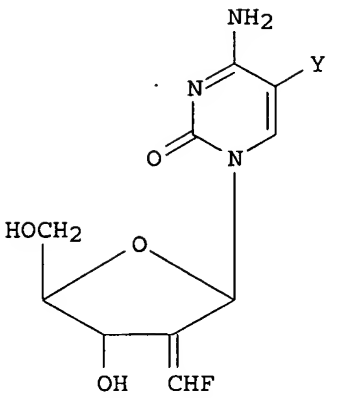


REFERENCE COUNT: 81 THERE ARE 81 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L97 ANSWER 36 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1996:231396 HCAPLUS
DOCUMENT NUMBER: 124:279153
TITLE: Treatment of neoplastic diseases by conjunctive therapy with 2'-fluoromethylene derivatives of pyridine deoxyribonucleosides and **radiation** or chemotherapy
INVENTOR(S): Snyder, Ronald D.
PATENT ASSIGNEE(S): Hoechst Marion Roussel, Inc., USA
SOURCE: PCT Int. Appl., 72 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 9601638	A1	19960125	WO 1995-US7205		19950506
W: AM, AT, AU, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LT, LU, LV, MD, MG, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TT					
RW: KE, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG					
AU 9527682	A1	19960209	AU 1995-27682		19950506
US 5595979	A	19970121	US 1995-495720		19950627
ZA 9505640	A	19960319	ZA 1995-5640		19950706
PRIORITY APPLN. INFO.:			US 1994-273242	A	19940711
			WO 1995-US7205	W	19950506

OTHER SOURCE(S): MARPAT 124:279153
GI



I

AB A patient afflicted with a neoplastic disease is administered an effective antineoplastic amount of ionizing or nonionizing **radiation**, or of a DNA-reactive chemotherapeutic agent in conjunction with a sensitizing amount of a title compound (I; V = O, CH2; Y = H, C1-4 alkyl, C1-4 alkoxy) or a salt thereof. Thus, 2'-deoxy-2'-fluoromethylenecytidine (I, V = O, Y = H) (II, 5 or 10 μM) radiosensitized HeLa cells to x-irradiation To prepare II, cytidine was protected at the 3' and 5' positions with 1,3-dichloro-1,1,3,3-tetraisopropyldisiloxane and at the NH2 group with DMF di-Me acetal, oxidized at 2' with oxalyl chloride and DMSO, condensed with FCH2SO2Ph in the presence of di-Et chlorophosphate and Li bis(trimethylsilyl)amide, deprotected at the NH2 group, converted with Bu3SnH and azobisisobutyronitrile to the fluoro(tributylstannyl)methylene derivative, and deprotected by refluxing in KF-MeOH.

IC ICM A61K031-70

CC 1-6 (Pharmacology)
Section cross-reference(s): 33

IT Deoxyribonucleic acids
RL: RCT (Reactant); RACT (Reactant or reagent)
(chemotherapeutic agents reaction with; combined treatment of neoplastic diseases with fluoromethylene derivs. of pyridine deoxyribonucleosides and **radiation** or chemotherapy)

IT Neoplasm inhibitors
Radiosensitizers, biological
(combined treatment of neoplastic diseases with fluoromethylene derivs. of pyridine deoxyribonucleosides and **radiation** or chemotherapy)

IT Neoplasm inhibitors
(carcinoma, combined treatment of neoplastic diseases with fluoromethylene derivs. of pyridine deoxyribonucleosides and **radiation** or chemotherapy)

IT Neoplasm inhibitors
(leukemia, combined treatment of neoplastic diseases with fluoromethylene derivs. of pyridine deoxyribonucleosides and **radiation** or chemotherapy)

IT Drug interactions
(synergistic, combined treatment of neoplastic diseases with fluoromethylene derivs. of pyridine deoxyribonucleosides and

radiation or chemotherapy)

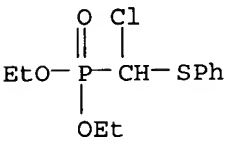
IT 130306-02-4P 171176-43-5P
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(combined treatment of neoplastic diseases with fluoromethylene derivs. of pyridine deoxyribonucleosides and **radiation** or chemotherapy)

IT 65-46-3, Cytidine 688-73-3, Tributyltin hydride 4637-24-5, Dimethylformamide dimethyl acetal 20808-12-2, Fluoromethyl phenyl sulfone **59664-75-4**, Diethyl 1-chloro-1-(phenylthio)methanephosphonate 69304-37-6, 1,3-Dichloro-1,1,3,3-tetraisopropyldisiloxane
RL: RCT (Reactant); RACT (Reactant or reagent)
(combined treatment of neoplastic diseases with fluoromethylene derivs. of pyridine deoxyribonucleosides and **radiation** or chemotherapy)

IT 90813-61-9P 114968-97-7P 149008-01-5P 149008-02-6P 149008-03-7P 149008-04-8P 149008-05-9P 149008-06-0P 149008-07-1P **153231-32-4P**
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(combined treatment of neoplastic diseases with fluoromethylene derivs. of pyridine deoxyribonucleosides and **radiation** or chemotherapy)

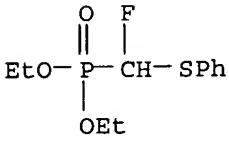
IT **59664-75-4**, Diethyl 1-chloro-1-(phenylthio)methanephosphonate
RL: RCT (Reactant); RACT (Reactant or reagent)
(combined treatment of neoplastic diseases with fluoromethylene derivs. of pyridine deoxyribonucleosides and **radiation** or chemotherapy)

RN 59664-75-4 HCAPLUS
CN Phosphonic acid, [chloro(phenylthio)methyl]-, diethyl ester (9CI) (CA INDEX NAME)



IT **153231-32-4P**
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(combined treatment of neoplastic diseases with fluoromethylene derivs. of pyridine deoxyribonucleosides and **radiation** or chemotherapy)

RN 153231-32-4 HCAPLUS
CN Phosphonic acid, [fluoro(phenylthio)methyl]-, diethyl ester (9CI) (CA INDEX NAME)



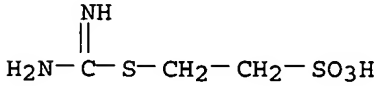
L97 ANSWER 37 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1996:628044 HCAPLUS
DOCUMENT NUMBER: 125:261148
TITLE: Processing of silver halide photographic materials
INVENTOR(S): Sugawa, Keiichi
PATENT ASSIGNEE(S): Konishiroku Photo Ind, Japan
SOURCE: Jpn. Kokai Tokkyo Koho, 23 pp.
CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 08190177	A2	19960723	JP 1995-1154	19950109
PRIORITY APPLN. INFO.:			JP 1995-1154	19950109

OTHER SOURCE(S): MARPAT 125:261148

AB The material is processed at following conditions; (1) using developer replenisher ≤250 mL/m2, (2) using fixer replenisher ≤350 mL/m2, and (3) using an automatic developer having a dry zone with a belt heated by a heat-conductive material at ≥90° or by heat-**radiating** material at ≥150°. The developer may contain ZSM [Z = alky, aromatic group, heterocycle, these are substituted for ≥1 of OH, SO3M1, COOM1, (substituted) amino, (substituted) ammonium; M1-2 = H, alkali metal, ammonium; M = H, alkali metal, ammonium, (substituted) amidino]. The method shows good film drying property even at low replenishing rate and gives clear images without background fog.

IC ICM G03C005-26
ICS G03C005-30; G03C005-31; G03C005-38; G03C005-395; G03C011-16
CC 74-2 (Radiation Chemistry, Photochemistry, and Photographic and Other Reprographic Processes)
IT 1074-36-8 2510-38-5 4695-30-1 15909-66-7 63684-27-5 63684-33-3 68994-94-5 **115750-75-9**
RL: MOA (Modifier or additive use); TEM (Technical or engineered material use); USES (Uses)
(silver sludge-preventing agent; photog. developer containing ascorbic acid derivative and mercapto or disulfide compound)
IT **115750-75-9**
RL: MOA (Modifier or additive use); TEM (Technical or engineered material use); USES (Uses)
(silver sludge-preventing agent; photog. developer containing ascorbic acid derivative and mercapto or disulfide compound)
RN 115750-75-9 HCAPLUS
CN Ethanesulfonic acid, 2-[(aminoiminomethyl)thio]-, monohydrochloride (9CI) (CA INDEX NAME)



● HCl

L97 ANSWER 38 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1995:667123 HCAPLUS
DOCUMENT NUMBER: 123:70398
TITLE: Heat mode recording and method for making a printing plate with it
INVENTOR(S): Verburgh, Yves; Dewanckele, Jean-Marie; Heugebaert, Franciscus; Leenders, Luc
PATENT ASSIGNEE(S): Agfa-Gevaert N. V., Belg.
SOURCE: Eur. Pat. Appl., 8 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 628409	A1	19941214	EP 1993-201686	19930611
EP 628409	B1	19970910		
R: BE, DE, FR, GB, NL				
PRIORITY APPLN. INFO.:			EP 1993-201686	19930611

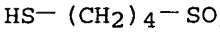
OTHER SOURCE(S): MARPAT 123:70398

AB A method for making a lithog. printing plate comprising image-wise exposing to actinic **radiation** a heat mode recording material comprising on a support a metallic layer and on top thereof a hydrophilic layer having a thickness of <50 nm thereby rendering the exposed areas hydrophobic and acceptant to greasy ink. The obtained printing plate may be used without further processing.

IC ICM B41C001-055
ICS B41N003-03; B41M005-24; B41N001-08; G03F007-07
CC 74-6 (Radiation Chemistry, Photochemistry, and Photographic and Other Reprographic Processes)
IT **84110-45-2**
RL: DEV (Device component use); USES (Uses)
(hydrophilizing agent; heat mode recording and method for making a printing plate with it)
IT **84110-45-2**
RL: DEV (Device component use); USES (Uses)
(hydrophilizing agent; heat mode recording and method for making a printing plate with it)
RN 84110-45-2 HCAPLUS
CN 1-Butanesulfonic acid, 4-mercapto-, compd. with guanidine (1:1) (9CI) (CA INDEX NAME)

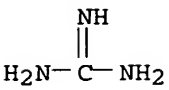
CM 1

CRN 24687-42-1
CMF C4 H10 O3 S2



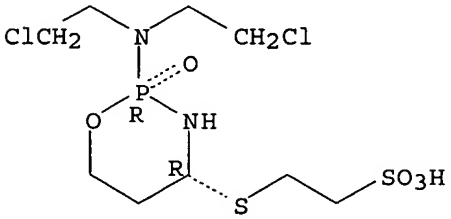
CM 2

CRN 113-00-8
CMF C H5 N3



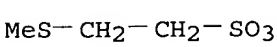
L97 ANSWER 39 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1996:122246 HCAPLUS
DOCUMENT NUMBER: 124:225686
TITLE: Evaluation of the protection of murine bone marrow stem cells by WR-2721 during in vitro treatment with mafosfamide
AUTHOR(S): Jiang, R.; Bony, V.; Lopez, M.
CORPORATE SOURCE: INSERM U76, C.N.T.S., Paris, Fr.
SOURCE: Progress in Clinical and Biological Research (1994), 389(Advances in Bone Marrow Purging and Processing), 23-9
CODEN: PCBRD2; ISSN: 0361-7742
PUBLISHER: Wiley-Liss
DOCUMENT TYPE: Journal
LANGUAGE: English
AB WR-2721 exhibited a significant protection of normal DBA/2 murine progenitor cells when bone marrow cells were exposed to mafosfamide (AsTA-Z) in vitro. However, when bone marrow cells were grafted into lethally **irradiated** mice, there was no difference in the survival rate of animals grafted whether or not the marrow was pre-incubated with WR-2721.
CC 9-11 (Biochemical Methods)
Section cross-reference(s): 1
IT **88859-04-5**, Mafosfamide
RL: ADV (Adverse effect, including toxicity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(protection of murine bone marrow stem cells by WR-2721 during in vitro treatment with mafosfamide)
IT **88859-04-5**, Mafosfamide
RL: ADV (Adverse effect, including toxicity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(protection of murine bone marrow stem cells by WR-2721 during in vitro treatment with mafosfamide)
RN 88859-04-5 HCAPLUS
CN Ethanesulfonic acid, 2-[[(2R,4R)-2-[bis(2-chloroethyl)amino]tetrahydro-2-oxido-2H-1,3,2-oxazaphosphorin-4-yl]thio]-, rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.



L97 ANSWER 40 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1995:286779 HCAPLUS
DOCUMENT NUMBER: 122:208505
TITLE: Methane as the product of reaction of

methyl-coenzyme-M with monovalent nickel complexes in aqueous solutions. A model for the in vivo activity of cofactor F430
AUTHOR(S): Zilbermann, Israel; Golub, Gilad; Cohen, Haim; Meyerstein, Dan
CORPORATE SOURCE: Nuclear Research Centre Negev, and Chemistry Department, Ben Gurion University of the Negev, Beer-Sheva, Israel
SOURCE: Inorganica Chimica Acta (1994), 227(1), 1-3
CODEN: ICHAA3; ISSN: 0020-1693
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Solns. containing the monovalent macrocyclic nickel complexes were prepared by **irradiating** with ionizing **radiation** He saturated solns. containing the divalent complexes and 0.01 M HCO2Na. Deaerated solns. containing methyl-coenzyme-M (MeCoM) were then injected into the vials containing the monovalent complexes. Vague traces of methane were detected at pH 7.4 while at pH 9.4 the yield of methane is over 10%. Blank expts. point out that MeCoM scavenges Me free radicals via a mechanism which does not produce methane as the major product. A mechanism for the formation of methane in these reactions is proposed.
CC 7-4 (Enzymes)
Section cross-reference(s): 29
IT 7440-02-0D, Nickel, complexes **53501-90-9**, Methyl-coenzyme-M 60182-60-7 131793-70-9
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); RCT (Reactant); BIOL (Biological study); PROC (Process); RACT (Reactant or reagent)
(methane as product of reaction of methyl-coenzyme-M with monovalent nickel complexes in aqueous solns. - a model for in vivo activity of cofactor F430)
IT **53501-90-9**, Methyl-coenzyme-M
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); RCT (Reactant); BIOL (Biological study); PROC (Process); RACT (Reactant or reagent)
(methane as product of reaction of methyl-coenzyme-M with monovalent nickel complexes in aqueous solns. - a model for in vivo activity of cofactor F430)
RN 53501-90-9 HCAPLUS
CN Ethanesulfonic acid, 2-(methylthio)- (9CI) (CA INDEX NAME)

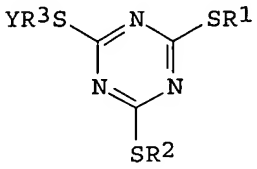


L97 ANSWER 41 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1994:232092 HCAPLUS
DOCUMENT NUMBER: 120:232092
TITLE: Negatively-working electrodeposition coating resin composition, electron deposition bath, and manufacture of resist pattern
INVENTOR(S): Amanokura, Hitoshi; Uehara, Hideaki; Tachiki, Shigeo; Kato, Takuro; Tsukada, Katsushige; Yamazaki, Juji; Takahashi, Tosha; Shiotani, Toshihiko; Nagashima, Yoshihisa
PATENT ASSIGNEE(S): Dai Nippon Toryo KK, Japan; Hitachi Chemical Co Ltd
SOURCE: Jpn. Kokai Tokkyo Koho, 8 pp.

CODEN: JKXXAF
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
JP 05281727	A2	19931029	JP 1992-77222	19920331
PRIORITY APPLN. INFO.:			JP 1992-77222	19920331

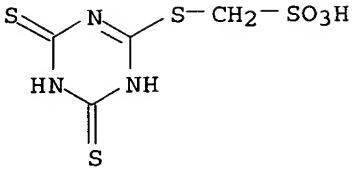
OTHER SOURCE(S): MARPAT 120:232092
GI



I

AB Claimed are (A) a neg.-working electrodeposition material containing (a) a polymer containing acrylic acid and/or methacrylic acid with acid value 20-300 neutralized by a basic organic compound, (b) water-insol. monomer including ≥2 photopolymerizable unsatd. linkages, (c) a water-insol. photopolymn. initiator, and (d) a triazine derivative I and/or its salt with a basic compound (Y = carboxyl, sulfonic acid group; R1-2 = H, alkyl; R3 = alkylene), (B) electrodeposition bath containing the composition, and (C) manufacture of resist pattern by a process including following successive steps; (1) impregnating an elec. conductive substrate as an anode in the bath, (2) forming an electrodeposited film under charging, (3) imagewise **irradiating** active beam to cure the exposed part, and (4) removing the unexposed part by developing. The process provides resist pattern showing no film residue after developing and high resolution

IC ICM G03F007-027
ICS C08F002-44; C08F002-50; C09D004-00; C09D005-44; C09D133-02; C25D013-06; G03F007-004; G03F007-028; G03F007-038; G03F007-30; H01L021-027; H05K003-00
ICA C25D013-00
CC 74-5 (Radiation Chemistry, Photochemistry, and Photographic and Other Reprographic Processes)
Section cross-reference(s): 38, 42
IT 154022-96-5 154022-97-6 **154022-98-7**
RL: USES (Uses)
((meth)acrylic acid copolymer composition containing, for electrodeposition coating, for neg.-working photoresist)
IT **154022-98-7**
RL: USES (Uses)
((meth)acrylic acid copolymer composition containing, for electrodeposition coating, for neg.-working photoresist)
RN 154022-98-7 HCAPLUS
CN Methanesulfonic acid, [(1,4,5,6-tetrahydro-4,6-dithioxo-1,3,5-triazin-2-yl)thio]- (9CI) (CA INDEX NAME)



L97 ANSWER 42 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1993:47948 HCAPLUS
DOCUMENT NUMBER: 118:47948
TITLE: Additives in the electrocrystallization process
AUTHOR(S): Plieth, W.
CORPORATE SOURCE: Inst. Phys. Chem., Freie Univ. Berlin, Berlin, D-1000/33, Germany
SOURCE: Electrochimica Acta (1992), 37(12), 2115-21
CODEN: ELCAAV; ISSN: 0013-4686
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Adding orgs. to a plating bath is the most frequently used method to achieve a special property of a metal layer. Several methods are in use for in situ control of their activity: cyclic voltammetry and laser light scattering are described in this paper. The concept of hard and soft acids and bases in discussed in order to understand competitive adsorption. More mol. information is obtained by spectroscopic methods. As an example, some recent results in monitoring silver deposition by surface enhanced Raman spectroscopy are described.
CC 72-8 (Electrochemistry)
Section cross-reference(s): 66, 73, 75
IT Laser **radiation**
(in electrocrystn.)
IT **64030-13-3**
RL: USES (Uses)
(in electrocrystn. of copper from bath containing poly(ethylene glycol))
IT **64030-13-3**
RL: USES (Uses)
(in electrocrystn. of copper from bath containing poly(ethylene glycol))
RN 64030-13-3 HCAPLUS
CN 1-Propanesulfonic acid, 3,3'-[1,2-ethanediylbis(thio)]bis-, disodium salt (9CI) (CA INDEX NAME)

$HO_3S-(CH_2)_3-S-CH_2-CH_2-S-(CH_2)_3-SO_3H$

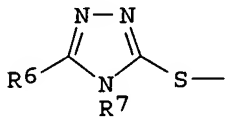
●2 Na

L97 ANSWER 43 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1989:553208 HCAPLUS
DOCUMENT NUMBER: 111:153208
TITLE: Preparation of disulfide-linked amphiphiles as drugs
INVENTOR(S): Roth, Hermann J.; Mueller, Christa E.
PATENT ASSIGNEE(S): Fed. Rep. Ger.
SOURCE: Ger. Offen., 7 pp.
CODEN: GWXXBX

DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 3728917	A1	19890309	DE 1987-3728917	19870829

PRIORITY APPLN. INFO.: DE 1987-3728917 19870829
OTHER SOURCE(S): MARPAT 111:153208
GI

Q= 

AB R1SSR2 [I; R1 = C8-20 alkyl, R3O2CCH2CHCO2R3; R2S = cysteine-, glutathione-, mercaptopurine-, N-acetylcysteine-, methylthiouracil-, propolythiouracil-residue, Q, etc.; R3 = R4CO2CH2CH(O2CR5)CH2, C8-20 alkyl; R4, R5 = C1-21 alkyl, cycloalkyl, Ph, PhCH2; R6 = H, CF3; R7 = H, Me] were prepared as antibacterial and antiviral agents, antineoplastics, radio- and liver-protective agents, etc. (no data). N-(Octadecylthio)phthalimide was refluxed 4.5 h with L-HSCH2CH(NH2)CO2H.HCl in EtOH to give 72% Me(CH2)17SSCH2CH(NH2)CO2H.

IC ICM C07C149-243
ICS C07C149-44; C07K005-02; C07D249-12; C07D473-38; C07D239-56; A61K045-05; A61K031-40; A61K031-195; A61K031-21; A61K031-505; A61K031-41

ICA C07C149-247

CC 23-9 (Aliphatic Compounds)
Section cross-reference(s): 1, 28, 34

ST amphiphile disulfide prepn drug; antiviral amphiphile prepn; antibacterial amphiphile prepn; antineoplastic amphiphile prepn; **radioprotective** amphiphile prepn; liver protective amphiphile prepn

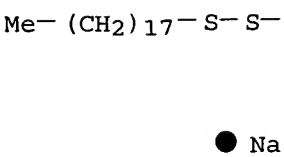
IT Bactericides, Disinfectants, and Antiseptics
Neoplasm inhibitors
Radioprotectants
Virucides and Virustats
(disulfide-linked amphiphiles)

IT 122504-71-6P 122504-74-9P 122504-75-0P 122504-83-0P 122504-88-5P
122714-63-0P 122714-64-1P 122714-65-2P 122714-66-3P
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of, as drug)

IT **122714-63-0P**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); SPN (Synthetic preparation); THU (Therapeutic use); BIOL (Biological study); PREP (Preparation); USES (Uses)
(preparation of, as drug)

RN 122714-63-0 HCAPLUS

CN Ethanesulfonic acid, 2-(octadecyldithio)-, sodium salt (9CI) (CA INDEX NAME)



L97 ANSWER 44 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1990:158882 HCAPLUS
DOCUMENT NUMBER: 112:158882
TITLE: Radical addition to vinyl phosphonates. A new synthesis of isosteric phosphonates and phosphonate analogs of α-amino acids
AUTHOR(S): Barton, Derek H. R.; Gero, Stephen D.; Quiclet-Sire, Beatrice; Samadi, Mohammad
CORPORATE SOURCE: Dep. Chem., Texas A and M Univ., College Station, TX, 77843, USA
SOURCE: Journal of the Chemical Society, Chemical Communications (1989), (15), 1000-1
CODEN: JCCCAT; ISSN: 0022-4936
DOCUMENT TYPE: Journal
LANGUAGE: English
OTHER SOURCE(S): CASREACT 112:158882

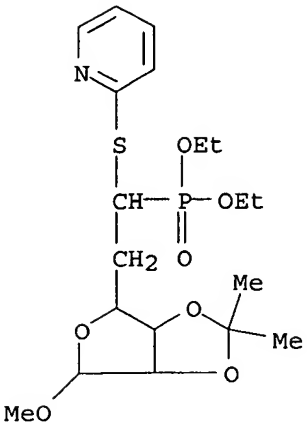
AB Derivs. of the phosphonic analogs of nucleotides and of side chain α-amino acids can be readily prepared by irradiation of acyl-N-hydroxy-2-thiopyridones in the presence of CH2:CHP(O)(OEt)2. Thus, Me3CO2CNHCH(CO2CH2Ph)CH2CH2CO2H was treated with ClCO2CH2CHMe2 and N-methylmorpholine followed by 2-mercaptopyridine 1-oxide. After 1 h, CH2:CHP(O)(OEt)2 was added and the mixture was **irradiated** with a tungsten lamp for 30 min at 0° to give 56 and 24% Me3CO2CNHCH(CO2CH2Ph)CH2CH2CH2CHRP(O)(OEt)2 (I, R = 2-pyridylthio, 2-thioxopyridin-1-yl, resp.). Reduction of the latter compds. with Bu3SnH and AIBN in PhH gave 76% I (R = H).

CC 34-2 (Amino Acids, Peptides, and Proteins)
Section cross-reference(s): 33

IT 119768-51-3P 119768-53-5P **125982-76-5P 125982-77-6P**
125982-79-8P 125982-80-1P 125982-83-4P 126004-12-4P
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation and reduction of)

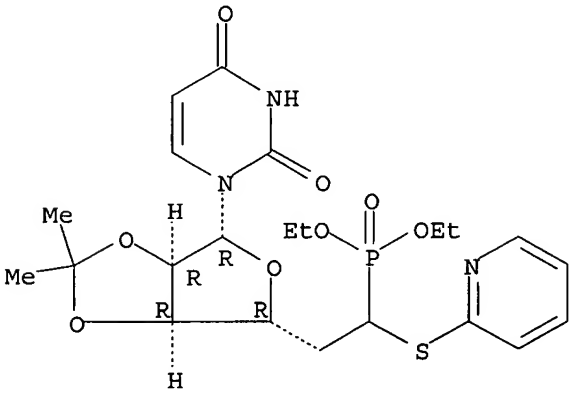
IT **125982-76-5P 125982-77-6P 125982-79-8P**
RL: RCT (Reactant); SPN (Synthetic preparation); PREP (Preparation); RACT (Reactant or reagent)
(preparation and reduction of)

RN 125982-76-5 HCAPLUS
CN β-D-ribo-Hexofuranoside, methyl 5-deoxy-6-C-(diethoxyphosphinyl)-2,3-O-(1-methylethylidene)-6-S-2-pyridinyl-6-thio- (9CI) (CA INDEX NAME)



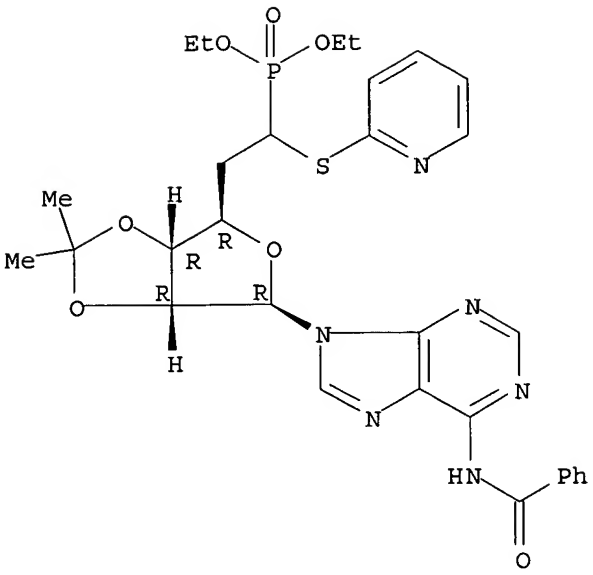
RN 125982-77-6 HCAPLUS
CN 2,4(1H,3H)-Pyrimidinedione, 1-[5-deoxy-6-C-(diethoxyphosphinyl)-2,3-O-(1-methylethylidene)-6-S-2-pyridinyl-6-thio-β-D-ribo-hexofuranosyl]- (9CI) (CA INDEX NAME)

Absolute stereochemistry.



RN 125982-79-8 HCAPLUS
CN Benzamide, N-[9-[5-deoxy-6-C-(diethoxyphosphinyl)-2,3-O-(1-methylethylidene)-6-S-2-pyridinyl-6-thio-β-D-ribo-hexofuranosyl]-9H-purin-6-yl]- (9CI) (CA INDEX NAME)

Absolute stereochemistry.

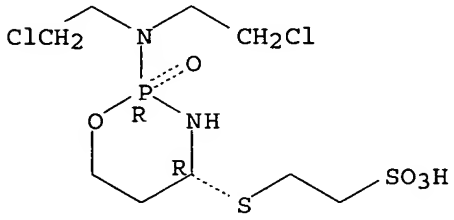


L97 ANSWER 45 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1988:622130 HCAPLUS
DOCUMENT NUMBER: 109:222130
TITLE: Ex vivo treatment of murine splenocyte-supplemented bone marrow inocula with mafosfamide prior to allogeneic transplantation in an attempt to prevent lethal graft-versus-host disease without compromising engraftment
AUTHOR(S): Kohn, Fred R.; Sladek, Norman E.
CORPORATE SOURCE: Dep. Pharmacol., Univ. Minnesota, Minneapolis, MN, 55455, USA
SOURCE: Immunopharmacology and Immunotoxicology (1988), 10(3), 387-98
CODEN: IITOEF; ISSN: 0892-3973
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Murine splenocyte-supplemented bone marrow cell suspensions were incubated with mafosfamide, an analog of "activated" cyclophosphamide, prior to transplantation across major histocompatibility barriers into lethally-**irradiated** recipient mice in an attempt to reduce the incidence of graft-vs.-host disease (GvHD)-related mortality without compromising engraftment. **Irradiated** mice that received vehicle-treated splenocyte-supplemented bone marrow inocula developed symptoms of severe GvHd and the majority of such animals did not survive. Treatment of donor cells with 160 μM mafosfamide for 30 min increased animal survival without evidence of GvHD. Survival of bone marrow allografts was demonstrated by the persistence of donor-type mononuclear cells in the peripheral blood of surviving animals. Treatment of donor cells with a four-fold higher concentration of mafosfamide also increased survival without evidence of GvHD; however, the host resistance to engraftment was indicated by a low percentage of donor mononuclear cells in the peripheral blood of survivors. Treatment of donor cells with a four-fold lower concentration of mafosfamide resulted in a slight increase in survival; however, all animals developed symptoms of GvHD. At appropriate concns., mafosfamide can eliminate GvHD-causing T lymphocytes from donor bone

marrow inocula without comprising its engraftment potential.
CC 1-7 (Pharmacology)
IT **88859-04-5**, Mafosfamide
RL: BIOL (Biological study)
(bone marrow and splenocyte transplant treatment by, graft rejection reduction by)
IT **88859-04-5**, Mafosfamide
RL: BIOL (Biological study)
(bone marrow and splenocyte transplant treatment by, graft rejection reduction by)
RN 88859-04-5 HCAPLUS
CN Ethanesulfonic acid, 2-[[(2R,4R)-2-[bis(2-chloroethyl)amino]tetrahydro-2-oxido-2H-1,3,2-oxazaphosphorin-4-yl]thio]-, rel- (9CI) (CA INDEX NAME)

Relative stereochemistry.

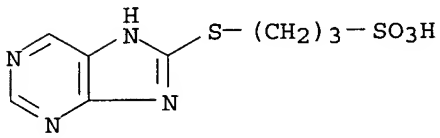


L97 ANSWER 46 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1986:621871 HCAPLUS
DOCUMENT NUMBER: 105:221871
TITLE: Relations between electronic and informational factors and the **radioprotective** effectiveness of sulfur-containing substances
AUTHOR(S): Mukhomorov, V. K.
CORPORATE SOURCE: S. M. Kirov Mil. Med. Acad., Leningrad, USSR
SOURCE: Radiobiologiya (1986), 26(4), 560-3
CODEN: RADOA8; ISSN: 0033-8192
DOCUMENT TYPE: Journal
LANGUAGE: Russian
AB The **radioprotective** activities of a number of S-containing compds. were analyzed in terms of the **radioprotective** information contained in their individual chemical constituents. A certain information threshold must be met before the substance is an effective **radioprotectant**.
CC 8-10 (Radiation Biochemistry)
ST **radioprotectant** sulfur compd structure information
IT Information, biological
(**radioprotectant**, of sulfur-containing compds.)
IT **Radioprotectants**
(sulfur-containing compds., structure-function relation of, chemical information in relation to)
IT Molecular structure-biological activity relationship
(**radioprotective**, of sulfur-containing compds.)
IT 52-66-4 638-43-7 694-59-7 758-28-1 1191-49-7 3687-18-1
3762-94-5 4378-70-5 4596-56-9 4621-66-3 6197-31-5 7250-31-9
7704-34-9D, compds. **10200-87-0** 10319-70-7 13338-50-6
13368-86-0 13441-72-0 13514-29-9 13551-09-2 18771-14-7
20537-88-6 20709-39-1 20724-76-9 **21668-81-5** 25452-97-5
29146-57-4 31098-42-7 34725-75-2 44744-78-7 44957-28-0
50433-21-1 54978-25-5 56235-27-9 56643-49-3 70548-43-5

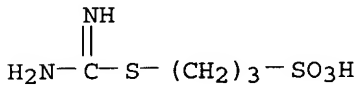
70548-45-7 78218-99-2 80085-11-6 82147-31-7 89034-17-3
90378-27-1 90378-29-3 90773-75-4 92046-25-8 93440-19-8
105289-99-4 105290-00-4 105290-01-5 105290-02-6 105290-03-7
105290-04-8 105290-05-9 105290-06-0 105290-08-2 105290-09-3
105290-10-6 105290-11-7 105290-12-8 105313-87-9
RL: PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**radioprotective** effectiveness of, structural information in relation to)

IT **10200-87-0 21668-81-5**
RL: PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**radioprotective** effectiveness of, structural information in relation to)

RN 10200-87-0 HCAPLUS
CN 1-Propanesulfonic acid, 3-(1H-purin-8-ylthio)- (9CI) (CA INDEX NAME)



RN 21668-81-5 HCAPLUS
CN 1-Propanesulfonic acid, 3-[(aminoiminomethyl)thio]- (9CI) (CA INDEX NAME)



L97 ANSWER 47 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1987:2365 HCAPLUS
DOCUMENT NUMBER: 106:2365
TITLE: Sulfur-35-labeled thio- and mercaptodiphosphonates as possible radiopharmaceuticals for the treatment of bone metastases
AUTHOR(S): Finck, W.; Unterspann, S.
CORPORATE SOURCE: Abt. Nuklearmed., Klin. Radiol., Rostock, DDR-2500, Ger. Dem. Rep.
SOURCE: Radioaktive Isotope in Klinik und Forschung (1986), 17(1), 469-74
CODEN: RIKFD7; ISSN: 0252-9440
DOCUMENT TYPE: Journal
LANGUAGE: German

AB Ethylmercaptomethanediphosphonate (I), methylmercaptopropan-2,5-diphosphonate (II), and diethyldimethylaminomethanethiodiphosphonate (III) formed complexes with 99mTc. Body retention of the Tc complexes after 6 h was .apprx.30, .apprx.20, and .apprx.30% for I, II, and III, resp.; .apprx.97% of the retained radioactivity was found in the skeleton. The complexes were accumulated at sites of bone repair at .apprx.10-fold higher concns. than in normal bones. Thus, the 35S-labeled compds., especially compound III (because of relatively easy preparation), could be used in the therapy of neoplastic metastases into bones.

CC 8-9 (Radiation Biochemistry)
Section cross-reference(s): 14

ST thiodiphosphonate sulfur 35 bone neoplasm; mercaptophosphonate sulfur 35 **radiotherapy** tumor

IT **Radiotherapy**
(of bone metastases, sulfur-35-labeled mercapto- and thiodiphosphonates metabolism in relation to)

IT Neoplasm
(**radiotherapy** of, sulfur-35-labeled mercapto- and thiodiphosphonate metabolism in relation to)

IT Bone, neoplasm
(**radiotherapy** of, sulfur-35-labeled mercapto- and thiodiphosphonates metabolism in relation to)

IT Blood
(sulfur-35-labeled mercapto- and thiodiphosphonates distribution in, bone metastases **radiotherapy** in relation to)

IT Kidney, metabolism
Liver, metabolism
Spleen, metabolism
(sulfur-35-labeled mercapto- and thiodiphosphonates metabolism by, bone metastases **radiotherapy** in relation to)

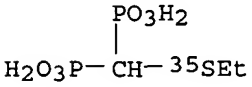
IT **105612-36-0 105612-37-1** 105612-38-2 105660-47-7
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(metabolism of, bone metastases **radiotherapy** in relation to)

IT 14133-76-7DP, alkylthiomethane diphosphonate derivative complexes **105612-39-3DP**, technetium-99m complexes 105612-40-6DP, technetium-99m complexes
RL: SPN (Synthetic preparation); PREP (Preparation)
(preparation and metabolism of metastable, bone metastases **radiotherapy** in relation to)

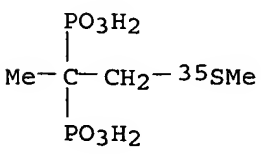
IT **67344-25-6DP**, technetium-99m complexes
RL: BPR (Biological process); BSU (Biological study, unclassified); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation); PROC (Process)
(preparation and metabolism of, bone metastases **radiotherapy** in relation to)

IT **105612-36-0 105612-37-1**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(metabolism of, bone metastases **radiotherapy** in relation to)

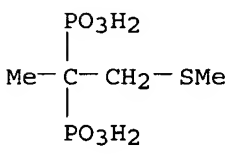
RN 105612-36-0 HCAPLUS
CN Phosphonic acid, [(ethylthio-35S)methylene]bis- (9CI) (CA INDEX NAME)



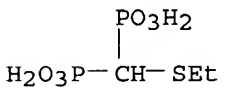
RN 105612-37-1 HCAPLUS
CN Phosphonic acid, [1-methyl-2-(methylthio-35S)ethylidene]bis- (9CI) (CA INDEX NAME)



IT **105612-39-3DP**, technetium-99m complexes
RL: SPN (Synthetic preparation); PREP (Preparation)
(preparation and metabolism of metastable, bone metastases **radiotherapy** in relation to)
RN 105612-39-3 HCAPLUS
CN Phosphonic acid, [1-methyl-2-(methylthio)ethylidene]bis- (9CI) (CA INDEX NAME)



IT **67344-25-6DP**, technetium-99m complexes
RL: BPR (Biological process); BSU (Biological study, unclassified); SPN (Synthetic preparation); BIOL (Biological study); PREP (Preparation); PROC (Process)
(preparation and metabolism of, bone metastases **radiotherapy** in relation to)
RN 67344-25-6 HCAPLUS
CN Phosphonic acid, [(ethylthio)methylene]bis- (9CI) (CA INDEX NAME)



L97 ANSWER 48 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1985:129728 HCAPLUS
DOCUMENT NUMBER: 102:129728
TITLE: Establishment, characterization, chemosensitivity, and radiosensitivity of two different cell lines derived from a human breast cancer biopsy
AUTHOR(S): Gioanni, Jeannine; Courdi, Adel; Lalanne, Claude Michel; Fischel, Jean Louis; Zanghellini, Evelyne; Lambert, Jean Claude; Ettore, Francette; Namer, Moise
CORPORATE SOURCE: Cent. Antoine Lacassagne, Nice, 06054, Fr.
SOURCE: Cancer Research (1985), 45(3), 1246-58
CODEN: CNREA8; ISSN: 0008-5472
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In vitro culture of a human breast cancer biopsy fragment gave rise to 2 permanent cell lines, CAL 18 A and CAL 18 B, which were differentiated by both morphol. and ultrastructural anal. The karyotypic and growth properties of these 2 cell lines also differed, providing further evidence of cell heterogeneity within a given tumor. Both cell lines lost their hormone receptors in vitro. CAL 18 A cells grew in agar and were tumorigenic after inoculation into nude mice; neither of these properties was observed in CAL 18 B cells. The chemosensitivity of 12 antineoplastic drugs was assessed by a short-term assay, using inhibition of tritiated thymidine incorporation by the cells after contact with the drugs as the end point. Only a few drugs were active at moderate concns. The overall responses of both cell lines were similar. The cell survival curves, established by the colony method following a single dose of **radiation**, were also very similar, despite the greater

heterogeneity of CAL 18 B cells. The 2 cell lines appear to be interrelated, since CAL 18 B cells were occasionally observed to emerge from CAL 18 A clones, suggesting that malignant cell redifferentiation may occur spontaneously in vitro.

CC 14-1 (Mammalian Pathological Biochemistry)
Section cross-reference(s): 1, 8

IT **Radiotherapy**
(mammary gland neoplasm-derived cell lines of human sensitivity to)

IT 51-21-8 57-22-7 59-05-2 147-94-4 148-82-3 865-21-4 1404-00-8 4375-07-9 15663-27-1 23214-92-8 29767-20-2 **84210-80-0**
RL: BIOL (Biological study)
(mammary gland tumor-derived cell lines of human sensitivity to)

IT **84210-80-0**
RL: BIOL (Biological study)
(mammary gland tumor-derived cell lines of human sensitivity to)

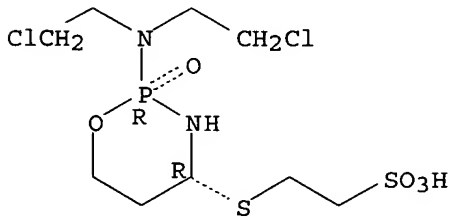
RN 84210-80-0 HCAPLUS

CN Ethanesulfonic acid, 2-[[(2R,4R)-2-[bis(2-chloroethyl)amino]tetrahydro-2-oxido-2H-1,3,2-oxazaphosphorin-4-yl]thio]-, rel-, compd. with cyclohexanamine (1:1) (9CI) (CA INDEX NAME)

CM 1

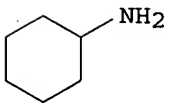
CRN 88859-04-5
CMF C9 H19 Cl2 N2 O5 P S2

Relative stereochemistry.



CM 2

CRN 108-91-8
CMF C6 H13 N



L97 ANSWER 49 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1985:89769 HCAPLUS
DOCUMENT NUMBER: 102:89769
TITLE: Effect of two cyclophosphamide derivatives on hemopoietic progenitor cells and pluripotential stem cells
AUTHOR(S): Porcellini, Adolfo; Manna, Annunziata; Talevi, Nadia; Sparaventi, Giovanni; Marchetti-Rossi, Maria Teresa; Baronciani, Donatella; De Biagi, Massimo

CORPORATE SOURCE: Div. Hematol., Hosp. Pesaro, Pesaro, Italy
SOURCE: Experimental Hematology (New York, NY, United States) (1984), 12(11), 863-6
CODEN: EXHMA6; ISSN: 0301-472X
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The effects of certain cyclophosphamide derivs. that have been used for selective removal of leukemic cells from marrow samples used for autologous transplantation were studied. The effect of in vitro treatment with 12-100 μg/mL of 4-hydroperoxycyclophosphamide (4-HC) [39800-16-3] and another cyclophosphamide congener, ASTA-Z 7557 [**84210-80-0**], on pluripotent stem cells (CFU-S) and committed progenitor cells (CFU-GM) in in bone marrow from mice was tested. The CFU-S were evaluated by the spleen colony assay at 8- and 12-days after transplant of the treated bone marrow cells into lethally **irradiated** mice. The 8-day colonies were transient in nature, rapidly growing, mainly erythroid, and lacked pluripotential precursors. The 12-day colonies provided a measure of hemopoietic stem cells as they slowly grew and contained primitive precursors. At the maximum dose levels tested, both drugs caused a 100% loss of CFU-GM and about 80%-95% inhibition of early transient CFU-S. In contrast, about 70% of the pluripotent 12-day CFU-S were spared. These data appear to explain the hemopoietic recovery seen in man after transplantation with marrow cells treated with 4-HC despite their relative absence of hemopoietic progenitor cells.

CC 1-6 (Pharmacology)

IT **Radiotherapy**
(of leukemia, cyclophosphamide derivs. effect on autologous transplantation of bone marrow in relation to)

IT 50-18-0D, derivs. 39800-16-3 **84210-80-0**
RL: BIOL (Biological study)
(hemopoietic progenitor cell and pluripotent stem cell response to, autologous transplantation of bone marrow in relation to)

IT **84210-80-0**
RL: BIOL (Biological study)
(hemopoietic progenitor cell and pluripotent stem cell response to, autologous transplantation of bone marrow in relation to)

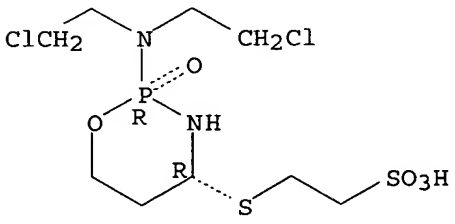
RN 84210-80-0 HCAPLUS

CN Ethanesulfonic acid, 2-[[(2R,4R)-2-[bis(2-chloroethyl)amino]tetrahydro-2-oxido-2H-1,3,2-oxazaphosphorin-4-yl]thio]-, rel-, compd. with cyclohexanamine (1:1) (9CI) (CA INDEX NAME)

CM 1

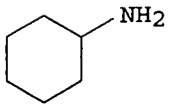
CRN 88859-04-5
CMF C9 H19 Cl2 N2 O5 P S2

Relative stereochemistry.



CM 2

CRN 108-91-8
CMF C6 H13 N



L97 ANSWER 50 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1984:112254 HCAPLUS
DOCUMENT NUMBER: 100:112254
TITLE: Aqueous processable, positive-working photopolymer compositions
INVENTOR(S): Proskow, Stephen
PATENT ASSIGNEE(S): du Pont de Nemours, E. I., and Co. , USA
SOURCE: U.S., 12 pp. Cont.-in-part of U.S. Ser. No. 271,411, abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

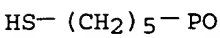
PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 4415651	A	19831115	US 1982-335051	19820104
EP 62474	A1	19821013	EP 1982-301644	19820329
EP 62474	B1	19850213		
R: BE, DE, FR, GB				
JP 57176035	A2	19821029	JP 1982-49218	19820329
JP 01039570	B4	19890822		
US 4415652	A	19831115	US 1982-400660	19820727

PRIORITY APPLN. INFO.: US 1981-271411 A2 19810330
US 1982-335051 A 19820104

AB Three-component aqueous processable, pos.-working photopolymer compns. containing an unsatd. polymer, a reactive mercapto acid, and a **radiation**-sensitive, radical-generating initiator are described for use as photoresists and photoimaging applications. Thus, a 20% solids coating solution containing an allyl methacrylate-Me methacrylate copolymer 1.00, mercaptosuccinic acid 0.150, ethylene diglycol caprylate 0.100, benzophenone 0.072, 4-methyl-2,6-di-tert-butylphenol 0.010, and CH2Cl2-MeOH (95:5) 5.328 g was coated on a Cu support to give a 25 μm thick film, imagewise exposed in a vacuum frame to a transparency, and then developed with an aqueous alkaline solution to give a well-defined pos. image.

IC G03C001-68
INCL 430277000
CC 74-4 (Radiation Chemistry, Photochemistry, and Photographic and Other Reprographic Processes)
IT 70-49-5 90-94-8 95-14-7 119-61-9, uses and miscellaneous 128-37-0, uses and miscellaneous 451-40-1 574-09-4 616-91-1 627-86-1 1707-68-2 2128-93-0 3274-12-2 3524-62-7 17689-17-7 22499-12-3 24650-42-8 26715-19-5 39088-65-8 51053-21-5 53802-03-2 63462-25-9 64111-86-0 69839-74-3 71310-21-9 84170-40-1 84214-67-5 84214-68-6 84214-69-7 84237-44-5 84297-16-5

84297-17-6 89022-61-7 89024-57-7
RL: USES (Uses)
(photoimaging compns. containing, pos.-working aqueous solution processable)
IT **84297-17-6**
RL: USES (Uses)
(photoimaging compns. containing, pos.-working aqueous solution processable)
RN 84297-17-6 HCAPLUS
CN Phosphonic acid, (5-mercaptopentyl)- (9CI) (CA INDEX NAME)



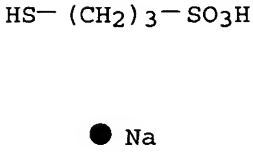
L97 ANSWER 51 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1979:444509 HCAPLUS
DOCUMENT NUMBER: 91:44509
TITLE: Pharmaceutically useful salts of mercaptoalkanesulfonic acids
INVENTOR(S): Brock, Norbert
PATENT ASSIGNEE(S): Asta-Werke A.-G. Chemische Fabrik, Fed. Rep. Ger.
SOURCE: Ger., 4 pp.
CODEN: GWXXAW
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 2756018	B1	19790329	DE 1977-2756018	19771214
DE 2756018	C2	19791122		
ZA 7806662	A	19791031	ZA 1978-6662	19781127
IL 56097	A1	19810913	IL 1978-56097	19781130
EP 2495	A1	19790627	EP 1978-101583	19781206
EP 2495	B1	19840215		
R: BE, CH, FR, GB, IT, LU, NL, SE				
AT 7808710	A	19800115	AT 1978-8710	19781206
AT 358162	B	19800825		
US 4220660	A	19800902	US 1978-967000	19781206
DK 7805539	A	19790615	DK 1978-5539	19781207
DK 154608	B	19881205		
DK 154608	C	19890508		
FI 7803756	A	19790615	FI 1978-3756	19781207
DD 140420	C	19800305	DD 1978-209691	19781212
DD 140420	B5	19950614		
CA 1117015	A1	19820126	CA 1978-317744	19781212
NO 7804192	A	19790615	NO 1978-4192	19781213
JP 54101432	A2	19790810	JP 1978-159066	19781213
JP 61054006	B4	19861120		
HU 22301	O	19820528	HU 1978-AA913	19781213
HU 179915	B	19830128		

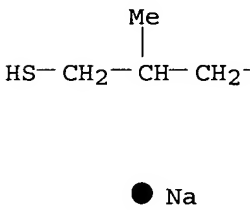
PRIORITY APPLN. INFO.: DE 1977-2756018 A 19771214
DE 1978-2827625 19780623

OTHER SOURCE(S): MARPAT 91:44509
AB Salts of HSXSO3H (X = C2-6 alkylene) are used to prevent cystitis in the treatment of cancer with alkylating agents. Thus, a patient treated with **radiation** therapy, 60 mg/kg ifosfamid, and 35 mg/kg Na 2-mercaptoethanesulfonate [19767-45-4] show no symptoms of hematurea.
IC A61K031-185; A61K031-095

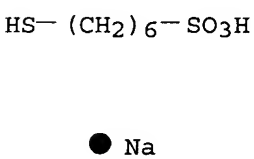
CC 63-6 (Pharmaceuticals)
IT **17636-10-1** 19767-45-4 **70793-14-5 70793-15-6**
RL: BIOL (Biological study)
(cystitis in cancer therapy treatment with)
IT **17636-10-1 70793-14-5 70793-15-6**
RL: BIOL (Biological study)
(cystitis in cancer therapy treatment with)
RN 17636-10-1 HCAPLUS
CN 1-Propanesulfonic acid, 3-mercapto-, monosodium salt (8CI, 9CI) (CA INDEX NAME)



RN 70793-14-5 HCAPLUS
CN 1-Propanesulfonic acid, 3-mercapto-2-methyl-, monosodium salt (9CI) (CA INDEX NAME)



RN 70793-15-6 HCAPLUS
CN 1-Hexanesulfonic acid, 6-mercapto-, monosodium salt (9CI) (CA INDEX NAME)



L97 ANSWER 52 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1980:400386 HCAPLUS
DOCUMENT NUMBER: 93:386
TITLE: Toxic and **radioprotective** properties of some phosphorus-containing isothiuronium derivatives
AUTHOR(S): Goloshchapova, Zh. A.
CORPORATE SOURCE: USSR
SOURCE: Trudy Instituta Ekologii Rastenii i Zhivotnykh, Ural'skii Nauchnyi Tsentr, Akademiya Nauk SSSR (1978), 113, 71-5
CODEN: TERZAP; ISSN: 0371-6473
DOCUMENT TYPE: Journal
LANGUAGE: Russian
AB Of the 27 isothiuronium derivs. tested, 18 showed lower acute toxicity in mice than did AET. Seven of the compds. showed **radioprotective**

activity and 2 of these, S-ethylisothiuronium metaphosphate [21704-44-9] and S-isopropylisothiuronium metaphosphate [73796-58-4] appeared to be as effective as AET in γ- **irradiated** mice. Increasing length of alkyl chains increased the toxicity in most cases. Substitution of the acetyl group with a benzoyl group increased the toxicity by >2-fold.

CC 1-3 (Pharmacodynamics)
Section cross-reference(s): 8

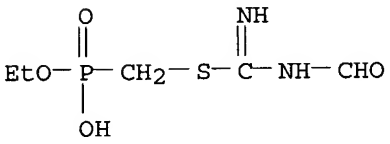
ST isothiuronium phosphorus deriv **radioprotectant**

IT **Radioprotectants**
(phosphorus-containing isothiuronium derivs. as)

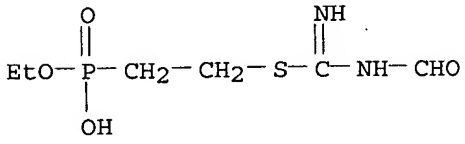
IT 538-28-3 16400-82-1 16400-83-2 16417-67-7 16417-83-7 16417-85-9
16417-86-0 21704-44-9 21704-46-1 25408-91-7 37031-92-8
37031-93-9 37031-95-1 55064-46-5 73794-26-0 73796-58-4
73796-59-5 73796-60-8 **73796-61-9** 73796-62-0 73796-63-1
73796-64-2 73796-65-3 73796-66-4 **73796-67-5**
73796-68-6
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(**radioprotective** activity of)

IT **73796-61-9 73796-64-2 73796-67-5**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(**radioprotective** activity of)

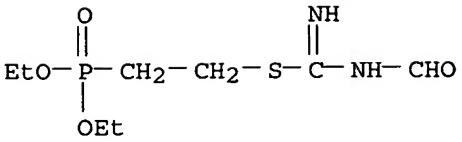
RN 73796-61-9 HCAPLUS
CN Carbamimidothioic acid, formyl-, (ethoxyhydroxyphosphinyl)methyl ester (9CI) (CA INDEX NAME)



RN 73796-64-2 HCAPLUS
CN Carbamimidothioic acid, formyl-, 2-(ethoxyhydroxyphosphinyl)ethyl ester (9CI) (CA INDEX NAME)



RN 73796-67-5 HCAPLUS
CN Carbamimidothioic acid, formyl-, 2-(diethoxyphosphinyl)ethyl ester, monohydrobromide (9CI) (CA INDEX NAME)



● HBr

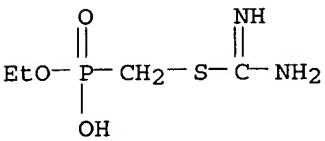
L97 ANSWER 53 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1977:594011 HCAPLUS
DOCUMENT NUMBER: 87:194011
TITLE: Effect of sulfur-containing **radioprotectants** on the evacuative function of the stomach in mice
AUTHOR(S): Grechka, I. I.; Zherebchenko, P. G.
CORPORATE SOURCE: USSR
SOURCE: Farmakologiya i Toksikologiya (Moscow) (1977), 40(5), 595-9
CODEN: FATOAO; ISSN: 0014-8318
DOCUMENT TYPE: Journal
LANGUAGE: Russian
AB The evacuative function of the mouse stomach was inhibited particularly by cysteamine-2HCl [56-17-7], aminopropylaminoethylthiophosphate [20537-88-6], aminopropylaminopropylthiophosphate [20709-39-1], bis(2-acetamidino) disulfide-2HCl [64632-46-8], bis(N-1-adamantylmethyl)-2-acetamidine disulfide-2HCl [37764-44-6], and N-(1-adamantylmethyl)-2-mercaptoacetamide-Hcl [22545-60-4]. Cystaphos [3724-89-8], 2-mercaptoacetamidine-HCl [19412-52-3], and cysteamine-HCl [156-57-0] were less effective and cysteamine bitartrate [27761-19-9] and disodium 4,4'-trithiobis(butane sulfonate) [**64632-45-7**] were least effective.
CC 1-5 (Pharmacodynamics)
ST **radioprotective** stomach evacuation
IT Stomach
(evacuation of, **radioprotectives** inhibition of)
IT **Radioprotectants**
(stomach evacuation inhibition by)
IT 56-17-7 156-57-0 3724-89-8 19412-52-3 20537-88-6 20709-39-1 22545-60-4 27761-19-9 37764-44-6 **64632-45-7** 64632-46-8
RL: BIOL (Biological study)
(stomach evacuation inhibition by)
IT **64632-45-7**
RL: BIOL (Biological study)
(stomach evacuation inhibition by)
RN 64632-45-7 HCAPLUS
CN 1-Butanesulfonic acid, 4,4'-trithiobis-, disodium salt (9CI) (CA INDEX NAME)

●2 Na

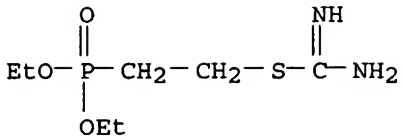
L97 ANSWER 54 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1976:173822 HCAPLUS
DOCUMENT NUMBER: 84:173822
TITLE: **Radioprotective** properties of some phosphorus-containing isothiuronium derivatives
AUTHOR(S): Il'yuchenok, T. Yu.; Frigidova, L. M.; Zav'yalov, Yu. V.; Verkhovskii, Yu. G.; Zherbin, E. A.; Shadurskii, K. S.; Mizrakh, L. I.; Polonskaya, L. Yu.
CORPORATE SOURCE: Lab. Radiats. Farmakol., Nauchno-Issled. Inst. Med. Radiol., Obninsk, USSR
SOURCE: Farmakologiya i Toksikologiya (Moscow) (1976), 39(2), 191-8
CODEN: FATOAO; ISSN: 0014-8318
DOCUMENT TYPE: Journal
LANGUAGE: Russian

AB Of 15 P-containing isothiuronium derivs. tested, only 1 had no **radioprotective** effects in γ- **irradiated** mice. Ethylphosphite S-ethylisothiuronium [16400-82-1], P,P'-diethylpyrophosphate bis-S-ethylthiuronium [37031-95-1],ethylene-bis-O-ethylphosphonate bis-S-ethylisothiuronium [37031-94-0], and O-ethyl-(chloromethyl)phosphonate S-ethylisothuronium [37031-93-9] given i.m. or i.p. were the most effective, increasing survival >50. Butylphosphite S-butylisothiuronium [16417-83-7] was the maximum toxic having an i.p. LD50 of 187 mg/kg; O-ethyl-diethylaminomethylphosphonate isothiuronium [59001-43-3] was the least toxic with an LD16 of 4200 mg/kg.

CC 1-5 (Pharmacodynamics)
ST isothiuronium phosphorus deriv **radioprotection** toxicity
IT **Radioprotectives**
(phosphorus-containing isothiuronium derivs.)
IT 1071-37-0 16400-82-1 16400-83-2 16417-83-7 21704-44-9 21704-46-1 25408-91-7 37031-93-9 37031-94-0 37031-95-1 **37031-96-2** 37031-97-3 39042-12-1 **51851-61-7** 59001-43-3
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(**radioprotection** by and toxicity of)
IT **37031-96-2 51851-61-7**
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(**radioprotection** by and toxicity of)
RN 37031-96-2 HCAPLUS
CN Carbamimidothioic acid, (ethoxyhydroxyphosphinyl)methyl ester (9CI) (CA INDEX NAME)



RN 51851-61-7 HCAPLUS
CN Carbamimidothioic acid, 2-(diethoxyphosphinyl)ethyl ester, monohydrobromide (9CI) (CA INDEX NAME)

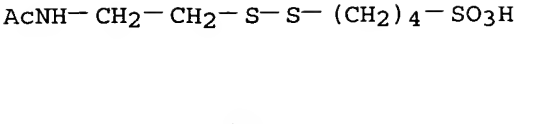


● HBr

L97 ANSWER 55 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1973:158919 HCAPLUS
DOCUMENT NUMBER: 78:158919
TITLE: Organic disulfide sulfinic acid compounds
INVENTOR(S): Field, Lamar; Barbee, Robert B.
PATENT ASSIGNEE(S): United States Dept. of the Army
SOURCE: U.S., 4 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 3723513	A	19730327	US 1969-874677	19691106
PRIORITY APPLN. INFO.:			US 1969-874677 A	19691106

GI For diagram(s), see printed CA Issue.
AB Heterocyclic disulfides (I, n = 3, 4, 5) were prepared by cyclization and and oxidation of alkanedithiols HS- (CH2)nSH; I (n = 4) was treated with H2NCH2CH2SH to give H2NCH2CH2S2(CH2)4S(O)OH (II) and with AcNHCH2CH2SH and NaOMe to give AcNHCH2CH2S2(CH2)4S(O)ONa (III). III was oxidized to the corresponding sulfonate (IV) with NaIO4. II, III, and IV were good **radioprotective** agents, with II giving the highest survival rates.
IC C07C
INCL 260513700
CC 23-12 (Aliphatic Compounds)
Section cross-reference(s): 28, 1
ST **radioprotective** butanesulfinate acetamidoethydithio aminoethyldithio; sulfinic acid aminoethyldithiobutane **radioprotective**; acetamidoethyldithiobutanesulfinate **radioprotective**; aminoethyldithiobutanesulfinic acid **radioprotective**
IT **Radioprotectives**
([[(aminoethyl)dithio]butanesulfinic acid and its acetamido and sulfonate derivs. as)
IT 18321-16-9P 18321-17-0P 19293-54-0P 19293-56-2P **19293-93-7P**
RL: SPN (Synthetic preparation); PREP (Preparation)
(preparation of)
IT **19293-93-7P**
RL: SPN (Synthetic preparation); PREP (Preparation)
(preparation of)
RN 19293-93-7 HCAPLUS
CN 1-Butanesulfonic acid, 4-[[2-(acetylamino)ethyl]dithio]-, monosodium salt (9CI) (CA INDEX NAME)



L97 ANSWER 56 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1971:546225 HCAPLUS
DOCUMENT NUMBER: 75:146225
TITLE: Photographic images by physical development of recording material with an exposed, **radiation** -sensitive layer consisting of organometallic compounds
INVENTOR(S): Bass, Jon D.
PATENT ASSIGNEE(S): Eastman Kodak Co.
SOURCE: Ger. Offen., 75 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 1949418	A	19701029	DE 1969-1949418	19690930
US 3647439	A	19720307	US 1968-764330	19681001
JP 51006535	B4	19760228	JP 1969-75902	19690925
FR 2019576	A5	19700703	FR 1969-33282	19690930
BE 739708	A	19700316	BE 1969-739708	19691001
GB 1292616	A	19721011	GB 1969-1292616	19691001
GB 1292617	A	19721011	GB 1969-1292617	19691001
PRIORITY APPLN. INFO.:			US 1968-764330 A	19681001
			US 1968-764332 A	19681001

AB Organosilver complexes with 0.2-5.0 mole organic compound/mole Ag and a pAg of 3-7, are used in a phys. development process in the proportion .apprx.30-200 mg Ag/ft2 support surface to give neutral images with high resolving power and opacity. Compds. of the following 8 types are utilized: (1) thioamides, thiazolinethiones, thiazolidinethiones, thiopyrimidines, oxazolidine-2-thiones, dithiocarbamates, or thioureas; (2) guanyl compds. such as pseudothiohydantoins, 2-thioimidazolidines, 2-thioimidazolines, or isothioureas; (3) mercapto acids, or mercaptoacetylamides; (4) alkynes; (5) hydroxyalkylcarboxylic acids or heterocyclic hydroxycarboxylic acids; (6) thiaalkyl compds.; (7) oxalic, phenylenedioxydialkylcarboxylic, or succinic acids; and (8) polymers with a ligand atom which can form a complex with the metal.
IC G03C
CC 74 (Radiation Chemistry, Photochemistry, and Photographic Processes)
IT 50-21-5D, Lactic acid, silver complexes 66-71-7D, 1,10-Phenanthroline, silver complexes 68-11-1D, Acetic acid, mercapto-, silver complexes 70-49-5D, Succinic acid, mercapto-, silver complexes 76-30-2D, Succinic acid, tetrahydroxy-, silver complexes 77-75-8D, 1-Pentyn-3-ol, 3-methyl-, silver complexes 77-92-9D, Citric acid, silver complexes 78-27-3D, Cyclohexanol, 1-ethynyl-, silver complexes 79-14-1D, Glycolic acid, silver complexes 81-07-2D, 1,2-Benzisothiazolin-3-one, 1,1-dioxide, silver complexes 87-69-4D, Tartaric acid, silver complexes 96-45-7D, 2-Imidazolidinethione, silver complexes 99-68-3D, Succinic acid, [(carboxymethyl)thio]-, silver complexes 102-39-6D, Acetic acid, (m-phenylenedioxy)di-, silver complexes 104-18-7D, Acetic acid,

[(p-aminophenyl)thio]-, silver complexes 105-31-7D, 1-Hexyn-3-ol, silver complexes 106-14-9D, Octadecanoic acid, 12-hydroxy-, silver complexes 107-54-0D, 1-Hexyn-3-ol, 3,5-dimethyl-, silver complexes 107-96-0D, Propionic acid, 3-mercapto-, silver complexes 111-17-1D, Propionic acid, 3,3'-thiodi-, silver complexes 115-19-5D, 3-Butyn-2-ol, 2-methyl-, silver complexes 119-80-2D, Benzoic acid, 2,2'-dithiodi-, silver complexes 123-93-3D, Acetic acid, thiodi-, silver complexes 135-13-7D, Benzoic acid, o-[(carboxymethyl)thio]-, silver complexes 141-90-2D, Uracil, 2-thio-, silver complexes 144-62-7D, Oxalic acid, silver complexes 148-24-3D, 8-Quinolinol, silver complexes 504-17-6D, Barbituric acid, 2-thio-, silver complexes 556-90-1D, 4-Thiazolidinone, 2-imino-, silver complexes 594-61-6D, Lactic acid, 2-methyl-, silver complexes 627-04-3D, Acetic acid, (ethylthio)-, silver complexes 922-67-8D, Propiolic acid, methyl ester, silver complexes 1073-72-9D, Phenol, p-(methylthio)-, silver complexes 1119-62-6D, Propionic acid, 3,3'-dithiodi-, silver complexes 1190-93-8D, Acetic acid, thio-, S-ester with mercaptoacetic acid, silver complexes 1934-75-4D, Dicyanamide, sodium salt, silver complexes 2068-24-8D, Acetic acid, (methylenedithio)di-, silver complexes 2295-31-0D, 2,4-Thiazolidinedione, silver complexes 4187-87-5D, Benzyl alcohol, α-ethynyl-, silver complexes 4265-54-7D, Acetic acid, (cyclohexylidenedithio)di-, silver complexes 4378-02-3D, 3-Butyn-2-ol, 2-cyclopropyl-, silver complexes 4822-44-0D, Acetanilide, 2-mercapto-, silver complexes 4938-00-5D, Propionic acid, 3-[(carboxymethyl)thio]-, silver complexes 5117-07-7D, 1H-Tetrazole, 5,5'-dithiobis[1-phenyl-, silver complexes 5217-47-0D, Barbituric acid, 1,3-diethyl-2-thio-, silver complexes 5398-29-8D, Propionic acid, 3-(amidinothio)-, silver complexes 6915-15-7D, Malic acid, silver complexes 7244-02-2D, Acetic acid, (ethylenedithio)di-, silver complexes 7404-50-4D, Acetic acid, (amidinothio)-, silver complexes 7440-22-4D, Silver, organic 7560-92-1D, 3-Cyclohexene-1-methanol, α-ethynyl-, silver complexes 10596-45-9D, 2-Propynylamine, N,N-diethyl-1-methyl-, silver complexes 15810-18-1D, Acetic acid, (ethylidenedithio)di-, silver complexes 15909-94-1D, Isophthalic acid, 5-(5-mercapto-1H-tetrazol-1-yl)-, silver complexes 16003-18-2D, Acetic acid, (2-furylthio)-, silver complexes 16003-20-6D, Propionic acid, 2-(2-furylthio)-, silver complexes 16003-21-7D, Propionic acid, 3-(2-furylthio)-, silver complexes 16003-22-8D, Succinic acid, (2-furylthio)-, silver complexes 16003-23-9D, Benzoic acid, o-(2-furylthio)-, silver complexes 16111-17-4D, Butyric acid, 3-(amidinothio)-, silver complexes 16945-88-3D, Benzoic acid, m-(4-methyl-2-thioxo-4-thiazolin-3-yl)-, silver complexes 16945-91-8D, 4-Thiazoline-3-succinic acid, 4-methyl-2-thioxo-, silver complexes 16945-92-9D, 4-Thiazoline-3-hexanoic acid, 4-methyl-2-thioxo-, silver complexes 16945-93-0D, 4-Thiazoline-3-butyric acid, 5-carboxy-4-methyl-2-thioxo-, silver complexes 16945-97-4D, 4-Thiazoline-3-butyric acid, 4-(carboxymethyl)-2-thioxo-, silver complexes 16946-00-2D, 4-Thiazoline-3-acetic acid, 5-acetyl-4-methyl-2-thioxo-, silver complexes 16946-01-3D, 4-Thiazoline-3-acetic acid, 5-carboxy-4-methyl-2-thioxo-, silver complexes 16946-04-6D, 4-Thiazoline-3,4-diacetic acid, 2-thioxo-, silver complexes 16946-05-7D, 4-Thiazoline-3,4-diacetic acid, 2-thioxo-, 4-ethyl ester, silver complexes 16946-34-2D, 4-Thiazoline-3-propionic acid, 4-methyl-2-thioxo-, silver complexes 16946-37-5D, 4-Thiazoline-3-propionic acid, 4-[(butylsulfonyl)methyl]-2-thioxo-, silver complexes 16946-39-7D, 4-Thiazoline-3-propionic acid, 5-acetyl-4-methyl-2-thioxo-, silver complexes 16946-41-1D, 4-Thiazoline-3-propionic acid, 5-carboxy-4-methyl-2-thioxo-, silver complexes 16946-43-3D, 4-Thiazoline-3-acetic acid, 4-carboxy-α-methyl-2-thioxo-, 4-ethyl ester, silver complexes 16946-44-4D, 4-Thiazoline-3-acetic acid, 4-carboxy-α-methyl-2-thioxo-, silver complexes 16946-45-5D,

4-Thiazoline-3,4-diacetic acid, α3-methyl-2-thioxo-, 4-ethyl ester, silver complexes 17356-19-3D, Cyclopentanol, 1-ethynyl-, silver complexes 20325-03-5D, Cyclohexanol, 4-tert-butyl-1-ethynyl-, silver complexes 20396-11-6D, Butyric acid, 4-(amidinothio)-, silver complexes 21153-31-1D, Propionic acid, 3,3'-thiobis[2,2-dimethyl-, silver complexes **21668-81-5D**, 1-Propanesulfonic acid, 3-(amidinothio)-, silver complexes 26473-47-2D, Propionic acid, 3-mercapto-2-methyl-, silver complexes 31090-12-7D, 4-Thiazoline-3-acetic acid, 4-methyl-2-thioxo-, silver complexes 31130-24-2D, 4-Thiazoline-3-butyric acid, 4-methyl-2-thioxo-, silver complexes 32386-31-5D, Acetic acid, (pentamethylenedithio)di-, silver complexes 41387-98-8D, 2-Oxazolidinethione, 5-methyl-, silver complexes 67427-11-6D, 1,3-Benzimidazolinedipropionic acid, 2-thioxo-, silver complexes 82720-22-7D, 3-Butyn-2-ol, 2-(p-chlorophenyl)-, silver complexes 91159-88-5D, 1H-Benzotriazole-5-sulfonic acid, silver complexes 99361-50-9D, Acetic acid, [(5-phenyl-1,3,4-oxadiazol-2-yl)thio]-, silver complexes 138687-60-2D, Hexanedioic acid, 2-mercapto-, silver complexes 357604-43-4D, Cyclohexanecarboxylic acid, 2-propynyl ester, silver complexes
RL: USES (Uses)
(photographic phys. development by)

IT **21668-81-5D**, 1-Propanesulfonic acid, 3-(amidinothio)-, silver complexes
RL: USES (Uses)
(photographic phys. development by)

RN 21668-81-5 HCAPLUS
CN 1-Propanesulfonic acid, 3-[(aminoiminomethyl)thio]- (9CI) (CA INDEX NAME)

L97 ANSWER 57 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1969:438878 HCAPLUS
DOCUMENT NUMBER: 71:38878
TITLE: Organic disulfides and related substances. XXVII. Reactions and synthetic utility of cyclic disulfides, dioxides, and tetroxides
AUTHOR(S): Field, Lamar; Barbee, Robert B.
CORPORATE SOURCE: Vanderbilt Univ., Nashville, TN, USA
SOURCE: Journal of Organic Chemistry (1969), 34(6), 1792-8
CODEN: JOCEAH; ISSN: 0022-3263
DOCUMENT TYPE: Journal
LANGUAGE: English
GI For diagram(s), see printed CA Issue.
AB Hydrolysis, polarographic reduction, and other reactions were studied of the unsubstituted 5-, 6-, and 7-membered disulfides, the 1,1-dioxides, and the 1,1,2,2-tetroxides. o-Dithiane 1,1,2,2-tetroxide (I) reacted less readily with PhSH than the 1,1-dioxide (II) but oxidized a thiolate quant. to the disulfide by a mild method of possible general use; its reactivity with nucleophiles resembled that of a disulfide, except for greater susceptibility to alkali (all 3 tetroxides were readily cleaved at pH 8); its pyrolysis gave tetrahydrothiophene dioxide but in low yield. Generalizations are difficult but seem usually to be for easier cleavage of the five-membered systems than of the six (with the seven variable) and for greater resistance to self-polymerization or to attack of a thiol by the
more

oxidized forms but for lesser resistance to hydrolysis and electrochem. reduction The dioxides and tetroxides are quite promising intermediates for synthesis. II underwent "oxodisulfide cleavage" by thiolate ion to give disulfides containing a sulfinate moiety, such as AcNHCH2CH2S2(CH2)4SO2Na (III) which in turn were converted into an alkyl or aryl sulfone, or a sulfonate. A typical disulfide product disproportionated to the sym. disulfides comparably to resistant classes, affording a synthesis of a disulfide-sulfone. I underwent "oxodisulfide cleavage" with alkali to give a sulfonate salt containing a sulfinate moiety, which was converted into a disulfide dioxide. III was active as an **antiradiation** drug.

CC 28 (Heterocyclic Compounds (More Than One Hetero Atom))

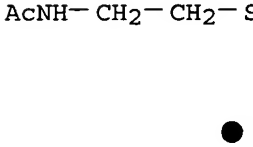
ST disulfides org polarographic redn; polarographic redn org disulfides; dithiane dioxides tetroxides **antiradiation** drugs

IT 18321-18-1P 19293-54-0P 19293-55-1P 19293-56-2P 19293-57-3P
19293-91-5P 19293-92-6P **19293-93-7P** 19293-94-8P
19293-95-9P 19293-96-0P 19293-97-1P
RL: SPN (Synthetic preparation); PREP (Preparation)
(preparation of)

IT **19293-93-7P**
RL: SPN (Synthetic preparation); PREP (Preparation)
(preparation of)

RN 19293-93-7 HCAPLUS

CN 1-Butanesulfonic acid, 4-[[2-(acetylamino)ethyl]dithio]-, monosodium salt (9CI) (CA INDEX NAME)



L97 ANSWER 58 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1966:508041 HCAPLUS
DOCUMENT NUMBER: 65:108041
ORIGINAL REFERENCE NO.: 65:20124g-h,20125a-b
TITLE: ω-(2-Mercaptoethylamino)-1-alkanesulfonic acid inner salts and related compounds as potential **antiradiation** agents
AUTHOR(S): Johnston, Thomas P.; Stringfellow, Carl R., Jr.
CORPORATE SOURCE: Kettering-Meyer Lab., Southern Res. Inst., Birmingham, AL
SOURCE: Journal of Medicinal Chemistry (1966), 9(6), 921-4
CODEN: JMCMAR; ISSN: 0022-2623
DOCUMENT TYPE: Journal
LANGUAGE: English

GI For diagram(s), see printed CA Issue.

AB Reactions of sultones Ia and Ib with cystamine, Na (S-2-aminoethyl)thiosulfate, and thiones such as thiosemicarbazide and pyridine-2(1H)-thione, provided a number of sulfonic acid inner salt derivs. for testing as **antiradiation** agents. Catalytic hydrogenolysis of the cystamine derivs. afforded ω-(2-mercaptoethylamino)-1-alkanesulfonic acids I and II. Sulfoalkylation products of 2-thiazolidinethione were observed to be particularly labile toward hydrolysis giving S-(2-aminoethyl) S'-(ω-sulfoalkyl) dithiocarbonates III and IV. 3,3'-[Dithiobis(ethylenimino)]bis(1-propanesulfonic acid) (V) and the derived thiol I showed good **radioprotective** activity in contrast to the inactivity of the

butane derivs. VI and II, the Bunte salts VII and VIII, and the dithiocarbonates III and IV. COH2)n-SOO2, HSCH2CH2N+H2(CH2)nSO3-, H3NCH2CH2SCOS(CH2)nSO3-; (Ia, n = 3), (I, n = 3), (III, n = 3); (Ib, n = 4), (II, r = 4), (IV, n = 4); [-SCH2CH2N+H2(CH2)nSO3-]z, -O3S(CH2)nN+H2CH2, H2SSO3-Na+; (V, n = 3), (VII, n = 3); (VI, n = 4), (VIII, n = 4); None, of, the isothiuronium-type sulfonates showed significant activity with the possible exception of 4-(acetimidoylthio)-1-butanesulfonic acid. 19 references.

CC 38 (Heterocyclic Compounds (More Than One Hetero Atom))

IT **Radiation** and **Radiation** effects
(protection against, ω-[(2-mercaptoethyl)amino]alkane sulfonic acid derivs. as)

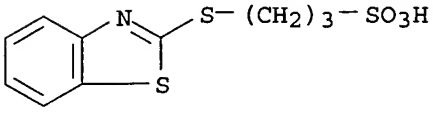
IT Sulfonic acids
(ω-[(2-mercaptoethyl)amino]alkane derivs., inner salts, as **antiradiation** agents)

IT 62-55-5, Acetimidic acid, thio-, esters, with 4-mercapto-1-butanesulfonic acid **4720-61-0**, 1-Propanesulfonic acid, 3-(2-benzothiazolylthio)- 7303-56-2, 7,8-Dithia-4,11-diazatetradecane-1,14-disulfonic acid 7303-57-3, 1-Propanesulfonic acid, 3-[(2-mercaptoethyl)amino]- 7303-58-4, 8,9-Dithia-5,12-diazahexadecane-1,16-disulfonic acid 7303-59-5, 1-Butanesulfonic acid, 4-[(2-mercaptoethyl)amino]- **7303-60-8**, Carbonic acid, dithio-, S-2-aminoethyl ester, S-ester with 3-mercapto-1-propanesulfonic acid **7303-61-9**, 1-Butanesulfonic acid, 4-mercapto-, S-ester with thioacetimidic acid 7308-43-2, Thiosulfuric acid, H2S2O3, S-ester with 3-[(2-mercaptoethyl)amino]-1-propanesulfonic acid, Na salt 7308-43-2, 1-Propanesulfonic acid, 3-[(2-mercaptoethyl)amino]-, hydrogen sulfate (ester), Na salt 7308-44-3, Thiosulfuric acid, H2S2O3, S-ester with 4-[(2-mercaptoethyl)amino]-1-butanesulfonic acid, Na salt 7308-44-3, 1-Butanesulfonic acid, 4-[(2-mercaptoethyl)amino]-, hydrogen sulfate (ester), Na salt **7313-50-0**, 1-Butanesulfonic acid, 4-mercapto-, S-ester with S-2-aminoethyl dithiocarbonate 7597-60-6, Formamide, N-(6-amino-1,2,3,4-tetrahydro-1,3-dimethyl-2,4-dioxo-5-pyrimidinyl)- 10184-00-6, Formamide, N-(6-amino-1,2,3,4-tetrahydro-2,4-dioxo-5-pyrimidinyl)- **10184-02-8**, 1-Propanesulfonic acid, 3-mercapto-, carbazimidate **10184-03-9**, 1-Butanesulfonic acid, 4-mercapto-, carbazimidate **10184-04-0**, 1-Propanesulfonic acid, 3-(2-thiazolin-2-ylthio)- **10200-80-3**, 1-Propanesulfonic acid, 3-(2-imidazolin-2-ylthio)- **10200-81-4**, 1-Butanesulfonic acid, 4-(2-imidazolin-2-ylthio)- **10200-82-5**, 1-Propanesulfonic acid, 3-(2-pyridylthio)- **10200-83-6**, 1-Butanesulfonic acid, 4-(2-pyridylthio)- **10200-84-7**, 1-Butanesulfonic acid, 4-(2-benzimidazolylthio)- **10200-86-9**, 1-Butanesulfonic acid, 4-(2-benzothiazolylthio)- **10200-87-0**, 1-Propanesulfonic acid, 3-(purin-8-ylthio)- **10200-88-1**, 1-Butanesulfonic acid, 4-(purin-8-ylthio)- **10250-26-7**, 1-Propanesulfonic acid, 3-(2-benzimidazolylthio)- 856620-54-7, Carbazimidic acid, thio-, ester with 3-mereapto-1-propanesulfonic acid
(preparation of)

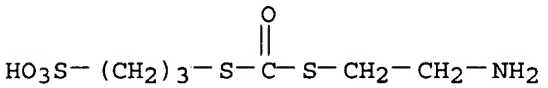
IT **4720-61-0**, 1-Propanesulfonic acid, 3-(2-benzothiazolylthio)- **7303-60-8**, Carbonic acid, dithio-, S-2-aminoethyl ester, S-ester with 3-mercapto-1-propanesulfonic acid **7303-61-9**, 1-Butanesulfonic acid, 4-mercapto-, S-ester with thioacetimidic acid **7313-50-0**, 1-Butanesulfonic acid, 4-mercapto-, S-ester with S-2-aminoethyl dithiocarbonate **10184-02-8**, 1-Propanesulfonic acid, 3-mercapto-, carbazimidate **10184-03-9**, 1-Butanesulfonic acid, 4-mercapto-, carbazimidate **10184-04-0**, 1-Propanesulfonic acid, 3-(2-thiazolin-2-ylthio)- **10200-80-3**, 1-Propanesulfonic acid, 3-(2-imidazolin-2-ylthio)- **10200-81-4**, 1-Butanesulfonic acid, 4-(2-imidazolin-2-ylthio)- **10200-82-5**, 1-Propanesulfonic

acid, 3-(2-pyridylthio)- **10200-83-6**, 1-Butanesulfonic acid,
4-(2-pyridylthio)- **10200-84-7**, 1-Butanesulfonic acid,
4-(2-benzimidazolylthio)- **10200-86-9**, 1-Butanesulfonic acid,
4-(2-benzothiazolylthio)- **10200-87-0**, 1-Propanesulfonic acid,
3-(purin-8-ylthio)- **10200-88-1**, 1-Butanesulfonic acid,
4-(purin-8-ylthio)- **10250-26-7**, 1-Propanesulfonic acid,
3-(2-benzimidazolylthio)-
(preparation of)

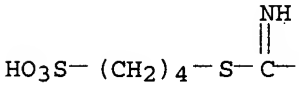
RN 4720-61-0 HCAPLUS
CN 1-Propanesulfonic acid, 3-(2-benzothiazolylthio)- (7CI, 8CI, 9CI) (CA INDEX NAME)



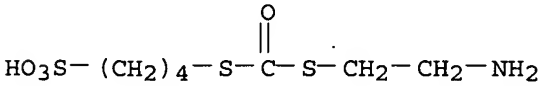
RN 7303-60-8 HCAPLUS
CN 1-Propanesulfonic acid, 3-[[[(2-aminoethyl)thio]carbonyl]thio]- (9CI) (CA INDEX NAME)



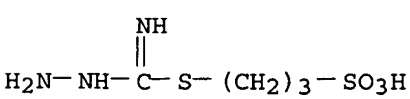
RN 7303-61-9 HCAPLUS
CN Ethanimidothioic acid, 4-sulfobutyl ester (9CI) (CA INDEX NAME)



RN 7313-50-0 HCAPLUS
CN 1-Butanesulfonic acid, 4-[[[(2-aminoethyl)thio]carbonyl]thio]- (9CI) (CA INDEX NAME)

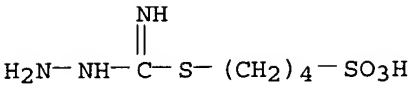


RN 10184-02-8 HCAPLUS
CN Carbazimidic acid, thio-, ester with 3-mercapto-1-propanesulfonic acid (7CI, 8CI) (CA INDEX NAME)

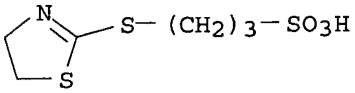


RN 10184-03-9 HCAPLUS
CN Carbazimidic acid, thio-, ester with 4-mercapto-1-butanesulfonic acid

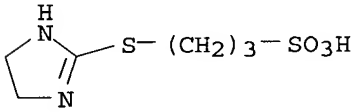
(7CI, 8CI) (CA INDEX NAME)



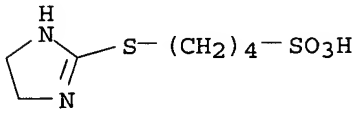
RN 10184-04-0 HCAPLUS
CN 1-Propanesulfonic acid, 3-(2-thiazolin-2-ylthio)- (7CI, 8CI) (CA INDEX NAME)



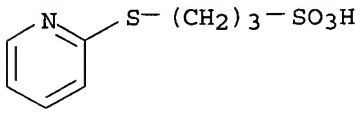
RN 10200-80-3 HCAPLUS
CN 1-Propanesulfonic acid, 3-(2-imidazolin-2-ylthio)- (6CI, 7CI, 8CI) (CA INDEX NAME)



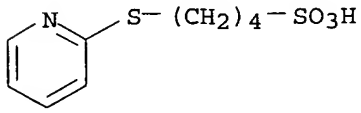
RN 10200-81-4 HCAPLUS
CN 1-Butanesulfonic acid, 4-(2-imidazolin-2-ylthio)- (7CI, 8CI) (CA INDEX NAME)



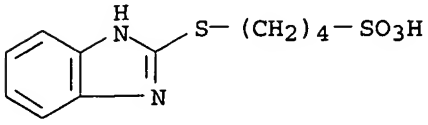
RN 10200-82-5 HCAPLUS
CN 1-Propanesulfonic acid, 3-(2-pyridylthio)- (7CI, 8CI) (CA INDEX NAME)



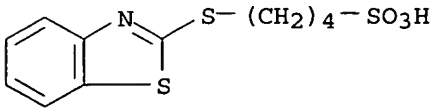
RN 10200-83-6 HCAPLUS
CN 1-Butanesulfonic acid, 4-(2-pyridylthio)- (7CI, 8CI) (CA INDEX NAME)



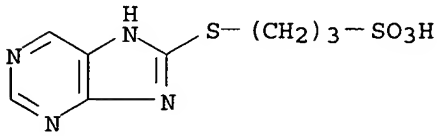
RN 10200-84-7 HCAPLUS
CN 1-Butanesulfonic acid, 4-(1H-benzimidazol-2-ylthio)- (9CI) (CA INDEX NAME)



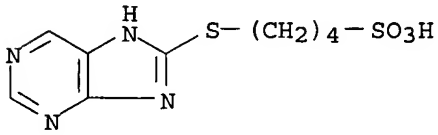
RN 10200-86-9 HCAPLUS
CN 1-Butanesulfonic acid, 4-(2-benzothiazolylthio)- (7CI, 8CI) (CA INDEX NAME)



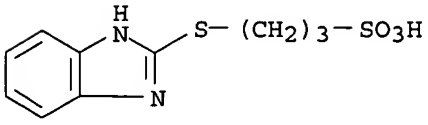
RN 10200-87-0 HCAPLUS
CN 1-Propanesulfonic acid, 3-(1H-purin-8-ylthio)- (9CI) (CA INDEX NAME)



RN 10200-88-1 HCAPLUS
CN 1-Butanesulfonic acid, 4-(purin-8-ylthio)- (7CI, 8CI) (CA INDEX NAME)



RN 10250-26-7 HCAPLUS
CN 1-Propanesulfonic acid, 3-(1H-benzimidazol-2-ylthio)- (9CI) (CA INDEX NAME)



L97 ANSWER 59 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1964:53256 HCAPLUS

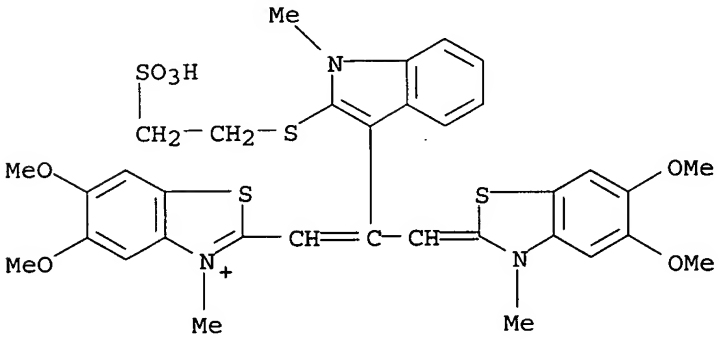
DOCUMENT NUMBER: 60:53256
ORIGINAL REFERENCE NO.: 60:9410c-g
TITLE: Nitrogenous condensation polymers, especially nylon, containing grafted acids
INVENTOR(S): Tanner, David
PATENT ASSIGNEE(S): E. I. du Pont de Nemours & Co.
SOURCE: 21 pp.
DOCUMENT TYPE: Patent
LANGUAGE: Unavailable
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 3099631		19630730	US 1958-719659	19580306
PRIORITY APPLN. INFO.:			US	19580306

AB Films, fabrics, and foams were prepared from a H2O-insol. graft copolymer comprising a high-mol.-weight linear nitrogenous condensation polymer, e.g. a polyamide, containing ≥300 titratable acid groups/106 g. polymer, at least 200 of the acid groups being chemical bonded by a C-to-C linkage to a catenarian C of the nitrogenous condensation polymer, the acid so linked being at least 1 C atom away from the catenarian C2. Thus, a swatch of nylon 66 fabric was padded to saturation with 25 g. maleic anhydride in 75 g. H2O, wrapped in Al foil and passed 40 times under an electron beam from a Van de Graaff electron accelerator (total exposure, 40 + 106 r.e.p.), removed from the Al foil and agitated for 2 hrs. in a 20 l. washer containing distilled H2O at 70°. The weight gain of the fabric after drying was 8%. The maleic acid-modified nylon was agitated for 2 hrs. in 18 l. distilled H2O containing 20 g. detergent and dried. An addnl. weight gain of 7% was noted. When hot ashes from a burning cigaret were flicked onto the fabric, only a small brown stain resulted. Holes were immediately melted through a fabric which had not undergone the above treatment. The treated fabric, when heated >185°, could be formed and drawn to 3 times its length at room temperature and also had a much drier handle than the untreated control. The maleic-acid modified fabric was soluble in 90% HCO2H but insol. in hot m-cresol. When the modified fabric was stirred for 1 hr. at 70° in 190 ml. distilled H2O containing 10 g. AcOH, it lost its high-temperature elastomeric properties, its hole-melting resistance was reduced, and it became soluble in hot m-cresol. In place of maleic acid, acrylic, itaconic, and fumaric acids could be used. Caprolactam, a polysulfonamide polymer, a poly(ether urethan), and an ether-ester-type polyurethan foam were similarly treated. A nylon 66 fabric was treated with an aqueous solution of K styrenesulfonate and, after **irradiation** with a dose of 15 + 106 r.e.p., had good antistatic properties. The sample was also resistant to hole-melting, was more resilient, and also more resistant to soiling than an untreated control.

INCL 260002500
CC 47 (Textiles)
IT Polymerization
(acid graft, on nitrogenous polymers by **radiation**)
IT Urethane polymers
(cellular, grafted by **radiation**, elec. charge-, hole melting- and soil-resistant)
IT Fibers, synthetic
(from nitrogen-containing polymers with grafts of acids linked to catenarian C by **irradiation**)
IT **Radiation** and **Radiation** effects
(polymerization (graft) by, of acids on nitrogenous polymers)

IT Nylon
(polymers (graft) with acrylic, fumaric, maleic or other acid group linked to catenarian C by **irradiation**)
IT Sulfonamides
(polymers of poly-, acid-grafted, γ- **irradiated**, dyeable, heat-resistant)
IT Electric charge
(prevention of, on nylon and nitrogenous polymers by grafting acids by **irradiation**)
IT 110-17-8, Fumaric acid
(polymers (graft) with nylon by **irradiation**, resistant to elec. charging, hole-melting and wet creasing)
IT **105070-08-4**, 2-[3-(5,6-Dimethoxy-3-methyl-2-benzothiazolinylidene)-2-[1-methyl-2-[(2-sulfoethyl)thio]indol-3-yl]propenyl]-5,6-dimethoxy-3-methylbenzothiazolium bromide
(preparation of)
IT 79-10-7, 2-Propenoic acid
(with nylon by **irradiation**, resistant to elec. charging, hole-melting and wet creasing)
IT **105070-08-4**, 2-[3-(5,6-Dimethoxy-3-methyl-2-benzothiazolinylidene)-2-[1-methyl-2-[(2-sulfoethyl)thio]indol-3-yl]propenyl]-5,6-dimethoxy-3-methylbenzothiazolium bromide
(preparation of)
RN 105070-08-4 HCAPLUS
CN 2-[3-(5,6-Dimethoxy-3-methyl-2-benzothiazolinylidene)-2-[1-methyl-2-[(2-sulfoethyl)thio]indol-3-yl]propenyl]-5,6-dimethoxy-3-methylbenzothiazolium bromide (7CI) (CA INDEX NAME)



● Br^-

L97 ANSWER 60 OF 90 HCAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1956:52721 HCAPLUS
DOCUMENT NUMBER: 50:52721
ORIGINAL REFERENCE NO.: 50:10124d-i,10125a
TITLE: Organophosphorus compounds
INVENTOR(S): Stiles, Alan R.; Rust, Frederick F.
PATENT ASSIGNEE(S): Shell Development Co.
DOCUMENT TYPE: Patent
LANGUAGE: Unavailable
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2742718		19551122	US	

AB The patent covers the preparation of compds. containing a C-P bond by addition of an olefinic substance and a compound with a H-P(O) grouping in the presence of free-radical initiators, such as organic peroxides. Thus, 0.4 mole NaH2PO2, 0.4 mole 1-octene, 100 ml. MeOH, and 0.0018 mole EtMeC(OOCMe3)2 heated in an autoclave 2 hrs. at 120° and diluted with H2O, gave a 100% conversion to soluble Na octylphosphinate. The use of 1-tetradecene similarly gave C14H29P(O)(H)ONa, which was analyzed. Similarly, (EtO)2POH or (BuO)2POH with 1-hexene, 1-octene, or 1-decene 16 hrs. at 120° in the presence of 5% (Me3CO)2 gave: 29% n-C6H13P(O)(OEt)2, b10 126°, nD20 1.4297, and 17.5% n-C6H13CHBuCH2P(O)(OEt)2, bhigh vac. 100°, nD20 1.4466; 21.2% n-C8H17P(O)(OBu)2, b1 146-9°, nD20 1.4394, n-C8H17CH(C6H13)CH2P(O)(OBu)2, bhigh vac. 100°, nD20 1.4463; 25.2% n-C10H21P(O)(OBu)2, b1 157°, nD20 1.4426, and 25.2% n-C10H21 CH(C8H17) CH2P(O)(OBu)2, bhigh vac. 155°, nD20 1.4533. Photoinitiation is also feasible: 1 mole 1-octene and 1 mole (BuO)2POH with 8.9% Me2CO **irradiated** with ultraviolet light 7 hrs. gave 54.5% n-C8H17P(O)(OBu)2, b1 146-52°, and a higher-boiling residue composed mainly of n-C8H17CH(C6H13)CH2P(O)(OBu)2. Heating 0.16 mole (CH2:CHCH2)2O, 0.32 mole (BuO)2 POH, and 4 mole-% (Me3CO)2 16 hrs. at 130° gave 28% mixed [(BuO)2P(O)CH2CH2CH2]2O and CH2:CHCH2OCH2CH2CH2P(O)(OBu)2, separated by mol. distillation from other products.

Similarly 0.15 mole MeCH:CHCO2Et, 0.3 mole (BuO)2POH, and 5 mole-% (Me3CO)2 gave 30% mixed (BuO)2P(O)CHMeCH2CO2Et and (BuO)2P(O)CHEtCO2Et, b1 135°. Similar reaction with CH2:CHCH2OH and (BuO)2POH gave 30% (BuO)2P(O)CH2CH2CH2OH, nD20 1.4478. CH2:CHCH2OH (0.15 mole), 0.3 mole (PRO)2POH, and 5 mole-% (Me3CO)2 gave about 30% (PrO)2P(O)CH2CH2CH2OH. (CH2:CHCH2S)2 and (BuO).2POH similarly gave [SCH2CH2CH2PO(OBu)2]2 and CH2:CHCH2SSCH2CH2CH2PO(OBu)2. Cyclohexene and (BuO)2POH gave some 40% (BuO)2P(O)C6H11, b1 134-40°, hydrolyzed with HCl to the free acid, m. 159-60°. Diallyl sulfide (1 mole) with 2 moles NaH2PO2 and 0.1 mole-% (Me3CO)2 in 8 hrs. at 120° gave CH2:CHCH2SCH2CH2CH2P(O)(H)ONa and S[CH2CH2CH2P(O)(H)ONa]2. (CH2:CH)2S and (EtO)2POH with 0.1 mole (CH2CO3CMe3)2 in 8 hrs. at 40° gave (EtO)2P(O)CH2CH2SCH:CH2 and S[CH2CH2P(O)(OEt)2]2. (CH2:CHS)2 and NH4H2PO2 with (Me3CO)2 in 8 hrs. at 120° gave [SCH2CH2P(O)(H)ONH4]2 and CH2:CHSSCH2CH2P(O)(H)ONH4. Olefins from petroleum cracking, containing 8-18 C atoms, heated similarly with (PrO)2POH in the presence of (Me3CO)2 16 hrs. at 130° gave mixed di-Pr alkylphosphonates. Similar reaction with olefins from cracked petroleum wax gave the corresponding alkylphosphinates in the form of Na salts. 1-Octene with PhP(O)(H)OEt and (Me3CO)2 in 17 hrs. at 130° gave 19% Ph(C8H17)P(O)OEt. NaH2PO2, 1-hexene, and MeEtC(OOCMe3)2 in MeOH heated 45 min. at 125° gave 0.4 mole C6H13P(O)(H)ONa; the resulting solution treated with an equivalent amount of 1-hexene and the catalyst and heated again 45 min. at 125° gave 60% (C6H13)2P(O)ONa.

CC 10 (Organic Chemistry)

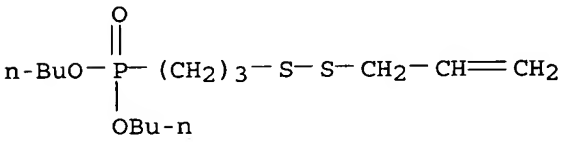
IT 1005-23-8, Phosphonic acid, cyclohexyl- 1085-92-3, Phosphonic acid, cyclohexyl-, dibutyl ester 4422-66-6, Butanoic acid, 3-phosphono- 5929-67-9, Phosphonic acid, octyl-, dibutyl ester 7681-53-0, Phosphinic acid, sodium salt 16165-66-5, Phosphonic acid, hexyl-, diethyl ester 17170-46-6, Phosphinic acid, hexyl-, sodium salt 36378-71-9, Phosphonic acid, decyl-, dibutyl ester 79252-46-3, Phosphinic acid, octyl-, sodium salt 88585-00-6, Phosphonic acid, (3-hydroxypropyl)-, dibutyl ester 95048-91-2, Phosphinic acid, tetradecyl-, sodium salt 109730-22-5,

Phosphinic acid, octylphenyl-, ethyl ester 111737-80-5, Phosphonic acid, (2-butyloctyl)-, diethyl ester **872813-15-5**, Phosphonic acid, [3-(allyldithio)propyl]-, dibutyl ester **872813-52-0**, Phosphonic acid, (thiodiethylene)di-, tetraethyl ester **872813-68-8**, Phosphonic acid, [dithiobis(trimethylene)]di-, tetrabutyl ester **872813-68-8**, 1-Propanephosphonic acid, 3,3'-dithiodi-, tetrabutyl ester **872813-85-9**, Phosphonic acid, (2-vinylthioethyl)-, diethyl ester 872818-40-1, Phosphonic acid, [oxybis(trimethylene)]di-, tetrabutyl ester 872818-40-1, 1-Propanephosphonic acid, 3,3'-oxydi-, tetrabutyl ester 872825-48-4, Phosphonic acid, 2-octyldodecyl-, dibutyl ester 873394-97-9, Phosphonic acid, [3-(allyloxy)propyl]-, dibutyl ester 878739-04-9, Phosphinic acid, [3-(allylthio)propyl]-, sodium salt
(preparation of)

IT **872813-15-5**, Phosphonic acid, [3-(allyldithio)propyl]-, dibutyl ester **872813-52-0**, Phosphonic acid, (thiodiethylene)di-, tetraethyl ester **872813-68-8**, Phosphonic acid, [dithiobis(trimethylene)]di-, tetrabutyl ester **872813-85-9**, Phosphonic acid, (2-vinylthioethyl)-, diethyl ester
(preparation of)

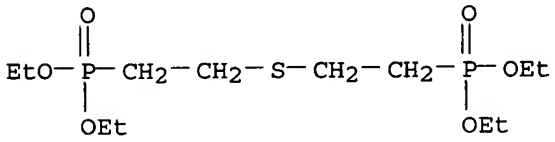
RN 872813-15-5 HCAPLUS

CN Phosphonic acid, [3-(allyldithio)propyl]-, dibutyl ester (5CI) (CA INDEX NAME)



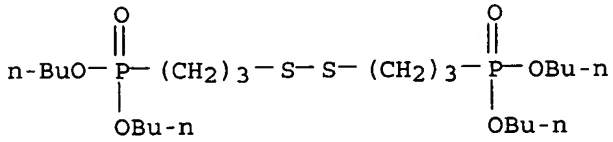
RN 872813-52-0 HCAPLUS

CN Phosphonic acid, (thiodiethylene)di-, tetraethyl ester (5CI) (CA INDEX NAME)



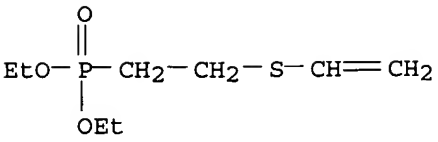
RN 872813-68-8 HCAPLUS

CN 1-Propanephosphonic acid, 3,3'-dithiodi-, tetrabutyl ester (5CI) (CA INDEX NAME)



RN 872813-85-9 HCAPLUS

CN Phosphonic acid, (2-vinylthioethyl)-, diethyl ester (5CI) (CA INDEX NAME)



L97 ANSWER 61 OF 90 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2002268661 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12008205
TITLE: BNP7787, a novel protector against platinum-related toxicities, does not affect the efficacy of cisplatin or carboplatin in human tumour xenografts.
AUTHOR: Boven E; Verschraagen M; Hulscher T M; Erkelens C A M; Hausheer F H; Pinedo H M; van der Vijgh W J F
CORPORATE SOURCE: Department of Medical Oncology, Vrije Universiteit Medical Centre, De Boelelaan 1117, Amsterdam, The Netherlands.. e.boven@vumc.edu
SOURCE: European journal of cancer (Oxford, England : 1990), (2002 May) Vol. 38, No. 8, pp. 1148-56.
Journal code: 9005373. ISSN: 0959-8049.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200207
ENTRY DATE: Entered STN: 15 May 2002
Last Updated on STN: 19 Jul 2002
Entered Medline: 18 Jul 2002

ABSTRACT:
BNP7787 (2',2'-dithio-bis-ethane sulphonate sodium), a water-soluble disulphide, is chemically and mechanistically different from other sulphur-containing chemoprotective agents. Presently, BNP7787 is under investigation for its protective properties with regard to the side-effects of platinum compounds. In this study, we evaluated BNP7787, mesna and amifostine for their effects on the antitumour activity of platinum compounds. Continuous exposure to BNP7787 did not affect the antiproliferative effects of cisplatin or carboplatin, but the efficacy of both compounds was reduced in the presence of mesna in vitro in two human ovarian cancer cell lines. BNP7787 or amifostine combined with cisplatin or carboplatin given in standard schedules for the treatment of nude mice bearing well-established OVCAR-3 xenografts did not interfere with platinum-induced inhibition of tumour growth. Of interest, BNP7787 or amifostine co-administered with carboplatin was significantly more effective than carboplatin alone ($P<0.01$). In the presence of amifostine, doses of cisplatin and carboplatin could be safely increased by factors of 1.6 and 1.5, respectively. Unlike in a previous study of BNP7787 in tumour-bearing rats, BNP7787 did not protect against additional weight loss following treatment with higher doses of cisplatin in OVCAR-3-bearing mice. Pharmacokinetics of (mixed) disulphides including BNP7787 and extractable mesna in deproteinised plasma revealed a rapid disappearance of BNP7787 and an AUC(5-60) value of mesna 9-fold lower than that calculated after an equivalent dose of mesna by weight. We can conclude that BNP7787 does not interfere with the antitumour activity of platinum compounds in vitro and in vivo. Clinical trials are underway to evaluate the protection of normal tissues by BNP7787 when combined with cisplatin.

CONTROLLED TERM: Check Tags: Female
Amifostine: PD, pharmacology
Animals
*Antineoplastic Agents: TU, therapeutic use
*Carboplatin: TU, therapeutic use
Cell Division: DE, drug effects
*Cisplatin: TU, therapeutic use
Drug Interactions
Humans
Lethal Dose 50
*Mesna: AA, analogs & derivatives
Mesna: BL, blood
Mesna: PK, pharmacokinetics
*Mesna: PD, pharmacology
Mice
Mice, Nude
Neoplasm Transplantation
*Ovarian Neoplasms: DT, drug therapy
Ovarian Neoplasms: PA, pathology
*Protective Agents: PD, pharmacology
Radiation-Protective Agents: PD, pharmacology
Transplantation, Heterologous
Weight Loss

CAS REGISTRY NO.: 15663-27-1 (Cisplatin); 19767-45-4 (Mesna); 20537-88-6 (Amifostine); 41575-94-4 (Carboplatin); **45127-11-5 (2,2'-dithiodiethanesulfonic acid)**

CHEMICAL NAME: 0 (Antineoplastic Agents); 0 (Protective Agents); 0 (**Radiation**-Protective Agents)

L97 ANSWER 62 OF 90 MEDLINE on STN DUPLICATE 2

ACCESSION NUMBER: 1998454919 MEDLINE

DOCUMENT NUMBER: PubMed ID: 9783598

TITLE: Modulation of platinum-induced toxicities and therapeutic index: mechanistic insights and first- and second-generation protecting agents.

AUTHOR: Hausheer F H; Kanter P; Cao S; Haridas K; Seetharamulu P; Reddy D; Petluru P; Zhao M; Murali D; Saxe J D; Yao S; Martinez N; Zukowski A; Rustum Y M

CORPORATE SOURCE: BioNumerik Pharmaceuticals, Inc, San Antonio, TX 78229, USA.

SOURCE: Seminars in oncology, (1998 Oct) Vol. 25, No. 5, pp. 584-99. Ref: 56
Journal code: 0420432. ISSN: 0093-7754.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199810

ENTRY DATE: Entered STN: 6 Jan 1999
Last Updated on STN: 6 Jan 1999
Entered Medline: 27 Oct 1998

ABSTRACT:
Platinum-type drugs have proven to be valuable in the treatment of a variety of solid tumors, beginning with the commercial approval of cisplatin 18 years ago. There are several clinically important toxicities commonly associated with the administration of these drugs. Despite the extensive use of cisplatin and carboplatin, the fundamental chemical transformations and mechanisms that underlie their antitumor and toxic effects have not been fully characterized. Several first-generation protective thiols have been clinically studied in an

attempt to reduce the toxicity of platinum-type drugs; while some of these agents appear to protect against certain toxicities, nearly all platinum-protecting drugs have their own intrinsic toxicities, which can be additive to the toxicity of platinum-type drugs. Tumor protection by platinum-protecting drugs is an additional untoward effect that is associated with certain types of agents and must be addressed with care. Recent advances in theoretical and laboratory methods and the use of supercomputers have extended our understanding of the possible major mechanisms underlying platinum drug antitumor activity and toxicity; we present strong evidence that there are two classes of chemical species of platinum drug. One class appears to predominantly account for the antitumor activity, and the other class of chemical species produces many of the toxic effects of platinum drugs. We have discovered a new nontoxic, second-generation platinum-protecting agent, known as BNP7787, which appears to selectively inactivate and eliminate toxic platinum species. BNP7787 has recently entered phase I clinical testing in cancer patients.

CONTROLLED TERM: ***Amifostine: PD, pharmacology**
Amifostine: TU, therapeutic use
Animals
*Antineoplastic Agents: AE, adverse effects
Antineoplastic Agents: CH, chemistry
Antineoplastic Agents: PD, pharmacology
Cisplatin: AE, adverse effects
Cisplatin: CH, chemistry
Cisplatin: PD, pharmacology
Drug Interactions
Humans
Kidney Diseases: CI, chemically induced
Kidney Diseases: PC, prevention & control
*Mesna: AA, analogs & derivatives
Mesna: PD, pharmacology
Mesna: TU, therapeutic use
*Platinum Compounds: AE, adverse effects
Platinum Compounds: CH, chemistry
Platinum Compounds: PD, pharmacology
Protective Agents: PD, pharmacology
Sulfhydryl Compounds: CH, chemistry
*Sulfhydryl Compounds: PD, pharmacology

CAS REGISTRY NO.: 15663-27-1 (Cisplatin); 19767-45-4 (Mesna); 20537-88-6 (Amifostine); **45127-11-5 (2,2'-dithiodiethanesulfonic acid)**

CHEMICAL NAME: 0 (Antineoplastic Agents); 0 (Platinum Compounds); 0 (Protective Agents); 0 (Sulfhydryl Compounds)

L97 ANSWER 63 OF 90 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 96156811 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8593069
TITLE: Isolation and identification of methanogen-specific DNA from blanket bog peat by PCR amplification and sequence analysis.
AUTHOR: Hales B A; Edwards C; Ritchie D A; Hall G; Pickup R W; Saunders J R
CORPORATE SOURCE: Department of Genetics and Microbiology, University of Liverpool, United Kingdom.
SOURCE: Applied and environmental microbiology, (1996 Feb) Vol. 62, No. 2, pp. 668-75.
Journal code: 7605801. ISSN: 0099-2240.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-L48407; GENBANK-L48408
ENTRY MONTH: 199604
ENTRY DATE: Entered STN: 18 Apr 1996
Last Updated on STN: 18 Apr 1996
Entered Medline: 1 Apr 1996

ABSTRACT:
The presence of methanogenic bacteria was assessed in peat and soil cores taken from upland moors. The sampling area was largely covered by blanket bog peat together with small areas of red-brown limestone and peaty gley. A 30-cm-deep core of each soil type was taken, and DNA was extracted from 5-cm transverse sections. Purified DNA was subjected to PCR amplification with primers IAf and 1100Ar, which specifically amplify 1.1 kb of the archaeal 16S rRNA gene, and ME1 and ME2, which were designed to amplify a 0.75-kb region of the alpha-subunit gene for **methyl coenzyme M** reductase (MCR). Amplification with both primer pairs was obtained only with DNA extracted from the two deepest sections of the blanket bog peat core. This is consistent with the notion that anaerobiosis is required for activity and survival of the methanogen population. PCR products from both amplifications were cloned, and the resulting transformants were screened with specific oligonucleotide probes internal to the MCR or archaeal 16S rRNA PCR product. Plasmid DNA was extracted from probe-positive clones of both types and the insert was sequenced. The DNA sequences of 8 MCR clones were identical, as were those of 16 of the 17 16S rRNA clones. One clone showed marked variation from the remainder in specific regions of the sequence. From a comparison of these two different 16S rRNA sequences, an oligonucleotide was synthesized that was 100% homologous to a sequence region of the first 16 clones but had six mismatches with the variant. This probe was used to screen primary populations of PCR clones, and all of those that were probe negative were checked for the presence of inserts, which were then sequenced. By using this strategy, further novel methanogen 16S rRNA variants were identified and analyzed. The sequences recovered from the peat formed two clusters on the end of long branches within the methanogen **radiation** that are distinct from each other. These cannot be placed directly with sequences from any cultured taxa for which sequence information is available.

CONTROLLED TERM: Base Sequence
DNA Primers: GE, genetics
DNA Probes: GE, genetics
*DNA, Bacterial: GE, genetics
*DNA, Bacterial: IP, isolation & purification
Ecosystem
*Euryarchaeota: GE, genetics
*Euryarchaeota: IP, isolation & purification
Genes, Bacterial
Genetic Markers
Molecular Sequence Data
Phylogeny
Polymerase Chain Reaction
RNA, Bacterial: GE, genetics
RNA, Ribosomal, 16S: GE, genetics
Research Support, Non-U.S. Gov't
Sequence Homology, Nucleic Acid
*Soil Microbiology

CHEMICAL NAME: 0 (DNA Primers); 0 (DNA Probes); 0 (DNA, Bacterial); 0 (Genetic Markers); 0 (RNA, Bacterial); 0 (RNA, Ribosomal, 16S)

L97 ANSWER 64 OF 90 MEDLINE on STN
ACCESSION NUMBER: 2004304500 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15206103

TITLE: The effect of cytoprotective agents in platinum anticancer therapy.
AUTHOR: Jakupec Michael A; Galanski Markus; Keppler Bernhard K
CORPORATE SOURCE: Institute of Inorganic Chemistry, University of Vienna, Waehringer Strasse 42, A-1090 Vienna, Austria.
SOURCE: Metal ions in biological systems, (2004) Vol. 42, pp. 179-208. Ref: 176
Journal code: 0406332. ISSN: 0161-5149.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200407
ENTRY DATE: Entered STN: 24 Jun 2004
Last Updated on STN: 13 Jul 2004
Entered Medline: 12 Jul 2004
CONTROLLED TERM: **Amifostine: TU, therapeutic use**
*Antineoplastic Agents: TU, therapeutic use
Antineoplastic Agents: TO, toxicity
*Cell Survival: DE, drug effects
Glutathione: TU, therapeutic use
Humans
*Mesna: AA, analogs & derivatives
Mesna: TU, therapeutic use
Neoplasms: DT, drug therapy
*Platinum Compounds: TU, therapeutic use
Platinum Compounds: TO, toxicity
*Protective Agents: TU, therapeutic use
Thioctic Acid: TU, therapeutic use
Vitamin E: TU, therapeutic use
CAS REGISTRY NO.: 1406-18-4 (Vitamin E); 19767-45-4 (Mesna); 20537-88-6 (Amifostine); **45127-11-5 (2,2'-dithiodiethanesulfonic acid)**; 62-46-4 (Thioctic Acid); 70-18-8 (Glutathione)
CHEMICAL NAME: 0 (Antineoplastic Agents); 0 (Platinum Compounds); 0 (Protective Agents)

L97 ANSWER 65 OF 90 MEDLINE on STN
ACCESSION NUMBER: 2001352046 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11414817
TITLE: Cryoreduction of **methyl-coenzyme M** reductase: EPR characterization of forms, MCR(ox1) and MCR (red1).
AUTHOR: Telser J; Davydov R; Horng Y C; Ragsdale S W; Hoffman B M
CORPORATE SOURCE: Department of Chemistry, Northwestern University, Evanston, Illinois 60208-3113, USA.
SOURCE: Journal of the American Chemical Society, (2001 Jun 27) Vol. 123, No. 25, pp. 5853-60.
Journal code: 7503056. ISSN: 0002-7863.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200109
ENTRY DATE: Entered STN: 10 Sep 2001
Last Updated on STN: 10 Sep 2001
Entered Medline: 6 Sep 2001
ABSTRACT:
Methyl-coenzyme M reductase (MCR) catalyzes the formation of **methyl-coenzyme M**

(CH(3)S-CH(2)CH(2)SO(3)) from methane. The active site is a nickel tetrahydrocorphinoid cofactor, factor 430, which in inactive form contains EPR-silent Ni(II). Two such forms, denoted MCR(silent) and MCR(ox1)(-)(silent), were previously structurally characterized by X-ray crystallography. We describe here the cryoreduction of both of these MCR forms by gamma-**irradiation** at 77 K, which yields reduced protein maintaining the structure of the oxidized starting material. Cryoreduction of MCR(silent) yields an EPR signal that strongly resembles that of MCR(red1), the active form of MCR; and stepwise annealing to 260-270 K leads to formation of MCR(red1). Cryoreduction of MCR(ox1)(-)(silent) solutions shows that our preparative method for this state yields enzyme that contains two major forms. One behaves similarly to MCR(silent), as shown by the observation that both of these forms give essentially the same redlike EPR signals upon cryoreduction, both of which give MCR(red1) upon annealing. The other form is assigned to the crystallographically characterized MCR(ox1)(-)(silent) and directly gives MCR(ox1) upon cryoreduction. X-band spectra of these cryoreduced samples, and of conventionally prepared MCR(red1) and MCR(ox1), all show resolved hyperfine splitting from four equivalent nitrogen ligands with coupling constants in agreement with those determined in previous EPR studies and from (14)N ENDOR of MCR(red1) and MCR(ox1). These experiments have confirmed that all EPR-visible forms of MCR contain Ni(I) and for the first time generated in vitro the EPR-visible, enzymatically active MCR(red1) and the activate-able "ready" MCR(ox1) from "silent" precursors. Because the solution Ni(II) species we assign as MCR(ox1)(-)(silent) gives as its primary cryoreduction product the Ni(I) state MCR(ox1), previous crystallographic data on MCR(ox1)(-)(silent) allow us to identify the exogenous axial ligand in MCR(ox1) as the thiolate from CoM; the cryoreduction experiments further allow us to propose possible axial ligands in MCR(red1). The availability of model compounds for MCR(red1) and MCR(ox1) also is discussed.

CONTROLLED TERM: Binding Sites
Coenzymes: CH, chemistry
Coenzymes: ME, metabolism
Electron Spin Resonance Spectroscopy: MT, methods
*Metalloporphyrins: CH, chemistry
Metalloporphyrins: ME, metabolism
*Methanobacteriales: EN, enzymology
Nickel: CH, chemistry
Nickel: ME, metabolism
Oxidation-Reduction
*Oxidoreductases: CH, chemistry
*Oxidoreductases: ME, metabolism
Research Support, U.S. Gov't, Non-P.H.S.

CAS REGISTRY NO.: 73145-13-8 (factor F430); 7440-02-0 (Nickel)

CHEMICAL NAME: 0 (Coenzymes); 0 (Metalloporphyrins); EC 1. (Oxidoreductases); EC 2.8.4.1 (**methyl coenzyme M** reductase)

L97 ANSWER 66 OF 90 MEDLINE on STN

ACCESSION NUMBER: 2000513863 MEDLINE

DOCUMENT NUMBER: PubMed ID: 11072815

TITLE: Methane formation by reaction of a methyl thioether with a photo-excited nickel thiolate--a process mimicking methanogenesis in archaea.

AUTHOR: Signor L; Knuppe C; Hug R; Schweizer B; Pfaltz A; Jaun B

CORPORATE SOURCE: Laboratorium fur Organische Chemie, Eidgenossische Technische Hochschule Zurich, Switzerland.

SOURCE: Chemistry (Weinheim an der Bergstrasse, Germany), (2000 Oct 2) Vol. 6, No. 19, pp. 3508-16.
Journal code: 9513783. ISSN: 0947-6539.

PUB. COUNTRY: GERMANY: Germany, Federal Republic of

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200011
ENTRY DATE: Entered STN: 22 Mar 2001
Last Updated on STN: 22 Mar 2001
Entered Medline: 28 Nov 2000

ABSTRACT:
The formation of a sulfuranyl radical intermediate followed by methyl transfer to the nickel(I) center of coenzyme F430 and generation of the disulfide has been proposed as a possible mechanism for the formation of methane catalyzed by ***methyl*** **coenzyme M** reductase in methanogenic archaea. In order to test this hypothesis, a sterically shielded, bifunctional model substrate that contained a methyl thioether and a sulfhydryl functional group, which could form a five-membered cyclic sulfuranyl radical according to the postulated mechanism, was synthesized. The corresponding thiolate reacted with Ni(II) salts to give a diamagnetic, square-planar Ni(II) dithiolate complex, which was characterized by X-ray diffraction. Upon ***irradiation*** of this complex with light of lambda > 300 nm, methane and the cyclic disulfide were formed, whereas **irradiation** of the thiolate in the absence of nickel gave only traces of methane and no cyclic disulfide. The observed products are consistent with the postulated mechanism via a sulfuranyl radical, and the role of light is interpreted as the formation of a Ni(I)/thiyl radical pair upon excitation of a charge-transfer band of the Ni(II) dithiolate. In the presence of a large excess of thiolate, the diamagnetic complex was transformed into a paramagnetic, five- or six-coordinate complex that proved to be more active in the generation of both methane and the cyclic disulfide, than the square-planar diamagnetic dithiolate.

CONTROLLED TERM: *Archaea: ME, metabolism
*Methane: ME, metabolism
*Nickel: CH, chemistry
Research Support, Non-U.S. Gov't
*Sulfhydryl Compounds: CH, chemistry
*Sulfides: CH, chemistry
CAS REGISTRY NO.: 74-82-8 (Methane); 7440-02-0 (Nickel)
CHEMICAL NAME: 0 (Sulfhydryl Compounds); 0 (Sulfides)

L97 ANSWER 67 OF 90 MEDLINE on STN
ACCESSION NUMBER: 1998438054 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9766664
TITLE: Reduction of dimesna to mesna by the isolated perfused rat liver.
AUTHOR: Goren M P; Hsu L C; Li J T
CORPORATE SOURCE: Department of Pathology and Laboratory Medicine, St. Jude Children's Research Hospital, Memphis, Tennessee 38105-2794, USA.
CONTRACT NUMBER: CA-21765 (NCI)
SOURCE: Cancer research, (1998 Oct 1) Vol. 58, No. 19, pp. 4358-62.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199811
ENTRY DATE: Entered STN: 6 Jan 1999
Last Updated on STN: 6 Jan 1999
Entered Medline: 3 Nov 1998

ABSTRACT:
Mesna is administered with ifosfamide and cyclophosphamide to reduce the

incidence of hemorrhagic cystitis. In the present model of mesna metabolism and disposition, mesna is rapidly and irreversibly oxidized to dimesna in the plasma, passes unchanged through the liver, and is then reduced by the kidney and excreted. Our detection of a high ratio of mesna to dimesna in the plasma of clinical samples led us to reinvestigate the hepatic metabolism of mesna and dimesna. We perfused isolated rat livers from female Sprague Dawley rats with protein-free buffered solution containing dimesna at concentrations observed during therapy. In single-pass perfusions, each liver was perfused with up to three dimesna concentrations during consecutive 20-min periods. Recirculating perfusions were used to study single supratherapeutic concentrations of dimesna or mesna. Mesna and dimesna concentrations were measured by specific chromatographic procedures. Dimesna reduction, adjusted by the effluent flow rate and liver weight (0.4-58.5 nmol/min/g liver), correlated closely by linear regression (r = 0.98; n = 36) to the perfused dimesna concentration (4.2-249 microM), indicating a clearance of 0.20 ml/min/g liver. The concentration of dimesna that entered the liver closely matched the summed concentration of mesna and dimesna emerging in the effluent perfusate (single-pass experiments: slope, 0.98; intercept, -0.30; r = 1.00; n = 31). Only trace amounts of unidentified thiols were detected in the bile during recirculation of perfusates with 1 mM mesna or 250 microM dimesna. The effluent mesna concentration correlated inversely with the flow rate, which was consistent with a low extraction ratio in the perfusion model. These data suggested that the dimesna reduction rate was limited by hepatic uptake. Dimesna reduction was decreased by agents that deplete glutathione. Pretreatment of rats with up to 100 mg/kg ifosfamide did not impair hepatic dimesna reduction. In control experiments, dimesna was not reduced during recirculation through the apparatus without a liver. Mesna was oxidized to dimesna during oxygenation of the perfusate in the reservoir, but mesna injected directly into the perfusate just before entry into the liver passed unchanged into the effluent. Extrapolation of the dimesna clearance data from the perfusion model to humans suggests that hepatic dimesna reduction may counterbalance the rapid oxidation of mesna in plasma. The proposed equilibrium is consistent with clinical observations and suggests a new model for mesna metabolism and disposition.

CONTROLLED TERM: Check Tags: Female
Animals
Biotransformation
Buthionine Sulfoximine: PD, pharmacology
Glutathione: ME, metabolism
In Vitro
Kinetics
Liver: DE, drug effects
*Liver: ME, metabolism
*Mesna: AA, analogs & derivatives
*Mesna: PK, pharmacokinetics
Oxidation-Reduction
Perfusion
Rats
Rats, Sprague-Dawley
Research Support, Non-U.S. Gov't
Research Support, U.S. Gov't, P.H.S.

CAS REGISTRY NO.: 19767-45-4 (Mesna); **45127-11-5 (2,2'-dithiodiethanesulfonic acid)**; 5072-26-4 (Buthionine Sulfoximine); 70-18-8 (Glutathione)

L97 ANSWER 68 OF 90 MEDLINE on STN
ACCESSION NUMBER: 91239514 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1903534
TITLE: Photoactivation of the 2-(**methylthio**) **ethanesulfonic acid** reductase from Methanobacterium.

AUTHOR: Olson K D; McMahon C W; Wolfe R S
CORPORATE SOURCE: Department of Microbiology, University of Illinois, Urbana 61801.
CONTRACT NUMBER: AI 12277 (NIAID)
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (1991 May 15) Vol. 88, No. 10, pp. 4099-103.
Journal code: 7505876. ISSN: 0027-8424.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199106
ENTRY DATE: Entered STN: 14 Jul 1991
Last Updated on STN: 3 Feb 1997
Entered Medline: 25 Jun 1991

ABSTRACT:
Inactive **2-(methylthio)ethanesulfonic** ***acid*** (CH3-S-CoM) reductase was partially activated by exposure to light. This simplified system replaces the complex enzymatic system of protein components A2, A3a, A3b, and ATP, which previously represented the only available means of reactivating the enzyme. Components necessary for light activation include N-(7-mercaptoheptanoyl)-L-threonine O3-phosphate (HS-HTP), CH3-S-CoM, titanium(III) citrate [Ti(III)Cit], and light above 400 nm. Photoactivation was inhibited by known inhibitors of methanogenesis: 2-bromoethanesulfonate (BES), N-(6-mercaptohexanoyl)-L-threonine O3-phosphate, N-(8-mercaptooctanoyl)-L-threonine O3-phosphate, and sodium dithionite. Methanogenesis continued when the light-activated reaction mixture was incubated in the dark. Although the specific activity was low (35 nmol of CH4 per h per mg of protein) the reaction products methane and the unsymmetrical disulfide of 2-mercaptoethanesulfonate (HS-CoM) and HS-HTP were identified. We were unable to photoactivate a reaction mixture containing the isolated prosthetic group, native F430, or its analogues.

CONTROLLED TERM:
Citrates: PD, pharmacology
Citric Acid
Disulfides: ME, metabolism
Enzyme Activation: RE, radiation effects
*Euryarchaeota: EN, enzymology
*Light
Mesna: AA, analogs & derivatives
Mesna: PD, pharmacology
Methane: ME, metabolism
*Oxidoreductases: ME, metabolism
Phosphothreonine: AA, analogs & derivatives
Phosphothreonine: PD, pharmacology
Photochemistry
Research Support, U.S. Gov't, Non-P.H.S.
Research Support, U.S. Gov't, P.H.S.

CAS REGISTRY NO.: 104302-77-4 (7-mercaptoheptanoylthreonine phosphate); 1114-81-4 (Phosphothreonine); 19767-45-4 (Mesna); **53501-90-9 (methyl coenzyme M)**; 74-82-8 (Methane); 77-92-9 (Citric Acid)
CHEMICAL NAME: 0 (Citrates); 0 (Disulfides); EC 1. (Oxidoreductases); EC 2.8.4.1 (**methyl coenzyme M** reductase)

L97 ANSWER 69 OF 90 MEDLINE on STN
ACCESSION NUMBER: 89291872 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2738065
TITLE: Coordination chemistry of F430. Axial ligation equilibrium

between square-planar and bis-aquo species in aqueous solution.

AUTHOR: Shiemke A K; Shelnutt J A; Scott R A
CORPORATE SOURCE: Department of Chemistry, University of Georgia, Athens 30602.

SOURCE: The Journal of biological chemistry, (1989 Jul 5) Vol. 264, No. 19, pp. 11236-45.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198908
ENTRY DATE: Entered STN: 9 Mar 1990
Last Updated on STN: 3 Feb 1997
Entered Medline: 2 Aug 1989

ABSTRACT:
X-ray absorption spectroscopic characterization of axial ligand coordination to factor F430, the nickel-tetrapyrrole cofactor of the S-**methyl**-***coenzyme*** **M** (CH3SCoM) methyl reductase enzyme from methanogenic bacteria, is presented. The nickel of isolated F430 is hexacoordinate at 10 K in aqueous solution (as is the enzyme-bound cofactor), whereas the epimerized and ring-oxidized derivatives of F430 have four-coordinate nickel. Reduction of the ring-oxidized derivative, F560, with dithionite yields F430 in its native configuration, with axial ligands indistinguishable from those present when the cofactor is obtained directly from the holoenzyme. Thus, we conclude that the axial ligands to F430 in aqueous solution are water molecules. Analysis of the nickel extended x-ray absorption fine structure is consistent with this conclusion. Resonance Raman spectra obtained at room temperature contain features characteristic of both 4- and 6-coordinate forms of the cofactor. We have found that the resonance Raman, optical, and x-ray absorption spectra of aqueous solutions of F430 are temperature-dependent due to a ligand-binding equilibrium involving the square-planar and 6-coordinate bis-aquo forms of the cofactor. At low temperatures (less than 250 K) the 6-coordinate form predominates, whereas higher temperature solutions contain both 4- and 6-coordinate species in a dynamic equilibrium. Similar behavior is observed in other weakly coordinating solvents such as methanol and ethanol. The 4-coordinate form is predominant in solvents with strong electron-withdrawing substituents such as 2,2,2-trifluoroethanol and 2-mercaptoethanol. The relevance of this facile ligand exchange to the active site structure and enzymatic mechanism of the parent enzyme is discussed.

CONTROLLED TERM:
Chemistry
Circular Dichroism
Coenzymes
Comparative Study
Euryarchaeota: EN, enzymology
Heat
Isomerism
Magnetic Resonance Spectroscopy
*Metalloporphyrins
*Metalloproteins
Molecular Structure
Multienzyme Complexes
*Nickel
Oxidation-Reduction
*Oxidoreductases: AN, analysis
Protein Conformation
Research Support, Non-U.S. Gov't
Research Support, U.S. Gov't, Non-P.H.S.

Solutions
Solvents
Spectrum Analysis
Spectrum Analysis, Raman
Structure-Activity Relationship
Thermodynamics
Water
X-Rays

CAS REGISTRY NO.: 73145-13-8 (factor F430); 7440-02-0 (Nickel); 7732-18-5 (Water)

CHEMICAL NAME: 0 (Coenzymes); 0 (Metalloporphyrins); 0 (Metalloproteins); 0 (Multienzyme Complexes); 0 (Solutions); 0 (Solvents); EC 1. (Oxidoreductases); EC 1.- (**methyl coenzyme M** methylreductase)

L97 ANSWER 70 OF 90 MEDLINE on STN
ACCESSION NUMBER: 88186873 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3356701
TITLE: Structural heterogeneity and purification of protein-free F430 from the cytoplasm of Methanobacterium thermoautotrophicum.
AUTHOR: Shiemke A K; Hamilton C L; Scott R A
CORPORATE SOURCE: School of Chemical Sciences, University of Illinois, Urbana 61801.
SOURCE: The Journal of biological chemistry, (1988 Apr 25) Vol. 263, No. 12, pp. 5611-6.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198805
ENTRY DATE: Entered STN: 8 Mar 1990
Last Updated on STN: 3 Feb 1997
Entered Medline: 18 May 1988

ABSTRACT:
F430 is the nickel containing tetrapyrrole cofactor of S-**methyl** ***coenzyme*** **M** methylreductase, the enzyme that catalyzes the final step of methane production by methanogenic bacteria: the reduction of S-***methyl*** **coenzyme M** (H3CSCH2CH2SO3-) to methane and coenzyme M (HSCH2CH2SO3-). The protein-free F430 obtained from the cytosol of Methanobacterium thermoautotrophicum, strain delta H, exists predominantly in two isomeric forms that differ in relative stereochemical disposition of acid side chains at the 12 and 13 positions of the macrocycle periphery (Pfaltz, A., Livingston, D. A., Jaun, B., Diekert, G., Thauer, R. K., and Eschenmoser, A. (1985) Helv. Chim. Acta 68, 1338-1358). A simple one-step chromatographic procedure for the large-scale separation of these isomers is described. X-ray absorption spectroscopic studies show that F430 (i.e. the native isomer) is 6-coordinate with long nickel-ligand bonds (approximately 2.1 A), suggesting an approximately planar macrocycle. In contrast, the 12,13-diepimer exhibits a 4-coordinate, square-planar structure with short nickel-nitrogen bonds (approximately 1.9 A), suggesting a ruffled macrocycle. Previous reports, based on other x-ray absorption spectroscopic data, of static disorder in F430 Ni-N distances are shown to be incorrect due to sample heterogeneity. The optical spectrum of F430 (whether purified from the protein-free cytosol or extracted at high ionic strength from the holoenzyme) differs significantly from that of the 12,13-diepimer. The optical spectral differences are correlated with the alterations in coordination number and geometry of the central nickel ion in the two F430 isomers.
CONTROLLED TERM: Chemistry, Physical

Chromatography, High Pressure Liquid
Chromatography, Ion Exchange
Coenzymes
Cytoplasm: AN, analysis
*Euryarchaeota: AN, analysis
*Metalloporphyrins
*Metalloproteins: IP, isolation & purification
Molecular Conformation
*Nickel: IP, isolation & purification
Research Support, Non-U.S. Gov't
Research Support, U.S. Gov't, Non-P.H.S.
Research Support, U.S. Gov't, P.H.S.
Spectrophotometry
Spectrum Analysis
X-Rays

CAS REGISTRY NO.: 73145-13-8 (factor F430); 7440-02-0 (Nickel)
CHEMICAL NAME: 0 (Coenzymes); 0 (Metalloporphyrins); 0 (Metalloproteins)

L97 ANSWER 71 OF 90 MEDLINE on STN
ACCESSION NUMBER: 87146979 MEDLINE
DOCUMENT NUMBER: PubMed ID: 2950482
TITLE: [Autograft of bone marrow treated by in vitro chemotherapy (Asta Z 7557) for consolidation of acute leukemia in adults in the first complete remission].
Autogreffe de moelle osseuse traitee par chimiotherapie in vitro (Asta Z 7557) en consolidation des leucemies aigues de l'adulte en premiere remission complete.
AUTHOR: Laporte J P; Gorin N C; Douay L; Lopez M; Najman A; Stachowiak J; Aegerter P; Lemonnier M P; Pene F; Kantor G; +
SOURCE: Presse medicale (Paris, France : 1983), (1987 Feb 28) Vol. 16, No. 7, pp. 338-42.
Journal code: 8302490. ISSN: 0755-4982.
PUB. COUNTRY: France
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: French
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198704
ENTRY DATE: Entered STN: 3 Mar 1990
Last Updated on STN: 3 Mar 1990
Entered Medline: 20 Apr 1987

ABSTRACT:
Fourteen adult patients in first complete remission of acute leukemia (A.L.) [6 with acute lymphoblastic leukemia (ALL), 8 with acute non lymphoblastic leukemia (ANLL)] were consolidated with high dose cyclophosphamide and total body **irradiation** followed by autologous bone marrow transplantation (ABMT) with marrow cleansed in vitro by Asta Z 7557. According to our previously described protocol showing evidence for a wide range of sensitivity from patient to patient, the marrow of each individual patient was incubated with the highest tolerable dose of Asta Z 7557. This dose, individually determined, was defined as the dose sparing between 0 and 10% of CFU-GM (CFU-GM DL95). ABMT was not followed by maintenance therapy. Hematological reconstitution was significantly faster in ALL patients when compared to ANLL patients. Out of these 14 patients: 2 relapsed on months 5 and 15 respectively after ABMT, and 2 died in complete remission on months 3 and 16 respectively, of veno-occlusive disease and encephalitis. Ten patients (70%) remain in complete remission up to a median of 15 months +, with 4 patients over 24 months +.

CONTROLLED TERM: Check Tags: Female; Male
Acute Disease

Adult
Bone Marrow: DE, drug effects
*Bone Marrow Transplantation
Combined Modality Therapy
Cyclophosphamide: AD, administration & dosage
*Cyclophosphamide: AA, analogs & derivatives
Cyclophosphamide: TU, therapeutic use
English Abstract
Humans
Leukemia: DT, drug therapy
*Leukemia: TH, therapy
Middle Aged
Research Support, Non-U.S. Gov't

CAS REGISTRY NO.: 50-18-0 (Cyclophosphamide); **88746-71-8 (Asta Z 7557)**

L97 ANSWER 72 OF 90 MEDLINE on STN
ACCESSION NUMBER: 86188218 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3516254
TITLE: Autologous bone marrow transplantation using marrow incubated with Asta Z 7557 in adult acute leukemia.
AUTHOR: Gorin N C; Douay L; Laporte J P; Lopez M; Mary J Y; Najman A; Salmon C; Aegerter P; Stachowiak J; David R; +
SOURCE: Blood, (1986 May) Vol. 67, No. 5, pp. 1367-76.
Journal code: 7603509. ISSN: 0006-4971.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198606
ENTRY DATE: Entered STN: 21 Mar 1990
Last Updated on STN: 21 Mar 1990
Entered Medline: 6 Jun 1986

ABSTRACT:
The sensitivity of human myeloblastic leukemic (CFU-L) and normal hemopoietic stem cells (CFU-GM and BFU-e) to Asta Z 7557 (INN Mafosfamide) was studied with regard to autologous bone marrow transplantation (ABMT) with cleansed marrow for consolidation therapy in adult patients with acute leukemia (AL) in remission. Establishment of the dose-response curves for CFU-GM (n = 37), BFUe (n = 11), and myeloblastic CFU-L (n = 9) demonstrated a wide range of sensitivity from patient to patient for all three progenitors. Whereas CFU-L, CFU-GM, and BFU-e grown in semisolid cultures disclosed similar sensitivities to Asta Z 7557, long-term culture (LTC) studies (n = 41) indicated a higher resistance of early progenitors. In an effort to achieve a maximum tumor cell kill and yet spare a sufficient amount of normal stem cells to ensure consistent engraftment, we defined the optimal dose for marrow cleansing as the dose sparing 5% CFU-GM (LD95). This dose was established from a preincubation test (PIT) realized on a 10-mL marrow aspirate taken 15 days before marrow collection in each individual patient. Twenty-four adult patients while in remission of AL (20 in complete remission, four in partial remission) were consolidated by cyclophosphamide 60 mg/kg X 2 and total body ***irradiation*** at 10 Gy followed by ABMT with marrow cleansed by Asta Z 7557 according to the specification described above. Patients were divided in two groups: group 1, unfavorable prognosis (11 patients); group 2, standard prognosis [13 patients in first complete remission (CR)]. All patients engrafted on leukocytes (median day for recovery to 10(9)/L: day 30), patients with ALL recovered faster than patients with ANL (median day 19 v 34). Similarly, recovery of platelets to 50.10(9)/L occurred sooner in patients with ALL (median day 67, range day 23 through 90) whereas three patients with acute

nonlymphoblastic leukemia (ANLL) in group 2 had to be supported with platelet transfusions for more than one year. In group 1, six patients had recurrent tumor within six months; three patients died from toxicity with no evidence of tumor. Two patients are still disease-free with a short follow-up (nine and ten months). In group 2, two patients died from toxicity with no evidence of leukemia three and 16 months post-ABMT. One patient with a M5 ANLL and one patient with ALL relapsed at six and 15 months, respectively. Nine patients have remained in CR or are disease-free with a median follow-up of 22 months.(ABSTRACT TRUNCATED AT 400 WORDS)

CONTROLLED TERM: Check Tags: Female; Male
Adolescent
Adult
*Bone Marrow Transplantation
Cell Separation
Clinical Trials
Colony-Forming Units Assay
*Cyclophosphamide: AA, analogs & derivatives
Cyclophosphamide: PD, pharmacology
Erythroblasts: CY, cytology
Granulocytes: CY, cytology
Humans
Leukemia: MO, mortality
*Leukemia: TH, therapy
Liver: PA, pathology
Middle Aged
Research Support, Non-U.S. Gov't
Stem Cells: CY, cytology
Stem Cells: DE, drug effects

CAS REGISTRY NO.: 50-18-0 (Cyclophosphamide); **88746-71-8 (Asta Z 7557)**

L97 ANSWER 73 OF 90 MEDLINE on STN
ACCESSION NUMBER: 86220596 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3519264
TITLE: Evaluation of lymphocyte subsets after autologous bone marrow transplantation with marrow treated by ASTA Z 7557 in acute leukemia: incidence of the in vitro treatment.
AUTHOR: Le Blanc G; Douay L; Laporte J P; Dominh A; Deloux J; Najman A; Duhamel G; Gorin N C
SOURCE: Experimental hematology, (1986 Jun) Vol. 14, No. 5, pp. 366-71.
Journal code: 0402313. ISSN: 0301-472X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198607
ENTRY DATE: Entered STN: 21 Mar 1990
Last Updated on STN: 21 Mar 1990
Entered Medline: 1 Jul 1986

ABSTRACT:
The lymphocyte subset reconstitution after high-dose chemotherapy and total body **irradiation** followed by autologous bone marrow transplantation (ABMT) has been studied in ten patients with acute leukemia (AL) (6 ALL and 4 ANLL) in complete remission (CR). Bone marrow was treated in vitro with high-dose ASTA Z 7557, individually determined according to CFU-GM sensitivity. The different peripheral blood lymphocyte subsets were characterized by means of monoclonal antibodies (indirect immunofluorescence assay) belonging to the following classes of differentiation: OKT11-T11 (CD2), OKT3-T3 (CD3), OKT4-T4 (CD4), OKT8-T8 (CD8), OKIa1-I2 (HLA-DR), Leu7 (natural killer/killer) and by

means of polyspecific antiimmunoglobulin sera (direct immunofluorescence assay). Data in these ten patients were compared with those of a control group of 21 normal donors and with a control group of 14 patients in CR without ABMT. Our results showed a marked depression of the T4:T8 ratio in patients with AL before ABMT, compared with normal donors who had respective values of 1.02 and 1.33 (p less than 0.01). This depression was increased and prolonged up to day 515 after ABMT, with a value of 0.32 (p less than 0.01 compared with the pregraft situation; p less than 0.001 compared with normal donors). This T4:T8 ratio imbalance was related to the depletion of the T4+ population and to the increase of the T8+ subset. This imbalance was emphasized after ABMT. The Leu 7+ population was also increased in grafted patients compared with normal donors (p less than 0.01). The B-cell population remained unchanged throughout the study. We conclude that patients autografted with marrow treated in vitro by high-dose ASTA Z 7557 may experience a long-term T-cell subset imbalance.

CONTROLLED TERM: Check Tags: Female; Male
Acute Disease
Adult
Bone Marrow: DE, drug effects
*Bone Marrow Transplantation
*Cyclophosphamide: AA, analogs & derivatives
Cyclophosphamide: PD, pharmacology
Humans
Killer Cells: CL, classification
Killer Cells, Natural: CL, classification
*Leukemia: TH, therapy
*Lymphocytes: CL, classification
T-Lymphocytes: CL, classification
Transplantation, Autologous

CAS REGISTRY NO.: 50-18-0 (Cyclophosphamide); **88746-71-8 (Asta Z 7557)**

L97 ANSWER 74 OF 90 MEDLINE on STN
ACCESSION NUMBER: 86190731 MEDLINE
DOCUMENT NUMBER: PubMed ID: 3516490
TITLE: The role of massive therapy with autologous bone marrow transplantation in Burkitt's lymphoma.
AUTHOR: Philip T; Pinkerton R; Hartmann O; Patte C; Philip I; Biron P; Favrot M
SOURCE: Clinics in haematology, (1986 Feb) Vol. 15, No. 1, pp. 205-17. Ref: 45
Journal code: 0331547. ISSN: 0308-2261.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198605
ENTRY DATE: Entered STN: 21 Mar 1990
Last Updated on STN: 6 Feb 1998
Entered Medline: 30 May 1986

ABSTRACT:
Burkitt's lymphoma has proved to be a very useful model for the evaluation of both massive therapy regimens and purging techniques. Results from several centres now confirm a number of general principles in relation to the use of ABMT procedures in this tumour. Patients in whom conventional chemotherapy has failed can be cured by massive therapy but this should be limited to those who have responded to salvage regimens or have only achieved first PR. Chemoresistant relapse is unlikely to be cured and the high probability of a transient response does not justify the procedure in such cases. Important ongoing clinical studies include the use of ABMT in first CR for CNS disease or

B-cell ALL. Results in allogeneic grafts suggest that current massive therapy regimens are curative in only 20-50% of patients (Appelbaum and Thomas, 1983) and new combinations are, therefore, still required. Phase I and II studies in patients with 'resistant relapse' are investigating the use of sequential high-dose alkylating agents and role of TBI. It is of particular importance to develop effective conventional 'salvage' regimens. Recent experience indicates that the combination of high-dose cisplatin and VP 16 is useful; other possibilities include high-dose interferon and high-dose cytarabine. Purging techniques in BL are now at an advanced stage and the combination of immunological and chemical treatments, once of proven efficacy in individual patients at a laboratory level, should be the subject of randomized studies.

CONTROLLED TERM: Antibodies, Monoclonal
Antineoplastic Agents: AD, administration & dosage
Antineoplastic Agents: AE, adverse effects
Antineoplastic Agents: TU, therapeutic use
Antineoplastic Combined Chemotherapy Protocols: AD, administration & dosage
Antineoplastic Combined Chemotherapy Protocols: AE, adverse effects
Antineoplastic Combined Chemotherapy Protocols: TU, therapeutic use
Bone Marrow: PA, pathology
*Bone Marrow Transplantation
Burkitt Lymphoma: CO, complications
Burkitt Lymphoma: DT, drug therapy
Burkitt Lymphoma: PA, pathology
Burkitt Lymphoma: RT, radiotherapy
*Burkitt Lymphoma: TH, therapy
Cell Separation: MT, methods
Central Nervous System Diseases: CO, complications
Child
Combined Modality Therapy
Complement System Proteins
Cyclophosphamide: AA, analogs & derivatives
Humans
Magnetics
Recurrence
Transplantation, Autologous
Whole-Body Irradiation: AE, adverse effects

CAS REGISTRY NO.: 50-18-0 (Cyclophosphamide); **88746-71-8 (Asta Z 7557)**; 9007-36-7 (Complement System Proteins)

CHEMICAL NAME: 0 (Antibodies, Monoclonal); 0 (Antineoplastic Agents)

L97 ANSWER 75 OF 90 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2005157399 EMBASE

TITLE: Prevention of chemotherapy-induced neuropathy: Leukemia inhibitory factor.

AUTHOR: Van Den Bent M.J.

CORPORATE SOURCE: M.J. Van Den Bent, Neuro-oncology Unit, Daniel Den Hoed Oncology Center, Erasmus University Medical Center, P.O. Box 5201, 3008AE Rotterdam, Netherlands. m.vandenbent@erasmusmc.nl

SOURCE: Clinical Cancer Research, (1 Mar 2005) Vol. 11, No. 5, pp. 1691-1693. .
Refs: 22
ISSN: 1078-0432 CODEN: CCREF4

COUNTRY: United States

DOCUMENT TYPE: Journal; Note

FILE SEGMENT: 008 Neurology and Neurosurgery

016 Cancer
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles

LANGUAGE: English

ENTRY DATE: Entered STN: 5 May 2005
Last Updated on STN: 5 May 2005

CONTROLLED TERM: Medical Descriptors:
*neuropathy: DT, drug therapy
*neuropathy: PC, prevention
*neuropathy: SI, side effect
*cancer chemotherapy
drug accumulation
peripheral neuropathy: SI, side effect
neurotoxicity: SI, side effect
sensorimotor neuropathy: SI, side effect
autonomic neuropathy: SI, side effect
sensory neuropathy: SI, side effect
dose response
nephrotoxicity: SI, side effect
bone marrow suppression: SI, side effect
drug effect
drug efficacy
drug potentiation
neuropathic pain: DR, drug resistance
neuropathic pain: SI, side effect
dysesthesia: SI, side effect
drug dose reduction
drug tolerability
side effect: SI, side effect
treatment outcome
paresthesia: SI, side effect
lung non small cell cancer: DT, drug therapy
human
nonhuman
clinical trial
note
priority journal
Drug Descriptors:
*antineoplastic agent: AE, adverse drug reaction
*antineoplastic agent: CT, clinical trial
*antineoplastic agent: CB, drug combination
*antineoplastic agent: DO, drug dose
*antineoplastic agent: IT, drug interaction
*antineoplastic agent: DT, drug therapy
*antineoplastic agent: PK, pharmacokinetics
*antineoplastic agent: PD, pharmacology
*leukemia inhibitory factor: CT, clinical trial
*leukemia inhibitory factor: DT, drug therapy
vincristine: AE, adverse drug reaction
cisplatin: AE, adverse drug reaction
cisplatin: CT, clinical trial
cisplatin: CB, drug combination
cisplatin: DO, drug dose
cisplatin: IT, drug interaction
cisplatin: DT, drug therapy
cisplatin: PK, pharmacokinetics
oxaliplatin: AE, adverse drug reaction
oxaliplatin: DO, drug dose
oxaliplatin: PD, pharmacology

paclitaxel: AE, adverse drug reaction
paclitaxel: CT, clinical trial
paclitaxel: CB, drug combination
paclitaxel: DO, drug dose
paclitaxel: DT, drug therapy
paclitaxel: PD, pharmacology
docetaxel: AE, adverse drug reaction
proteasome inhibitor: AE, adverse drug reaction
proteasome inhibitor: DO, drug dose
bortezomib: AE, adverse drug reaction
bortezomib: DO, drug dose
Vinca alkaloid: AE, adverse drug reaction
carboplatin: AE, adverse drug reaction
carboplatin: CB, drug combination
carboplatin: PD, pharmacology
taxane derivative: AE, adverse drug reaction
taxane derivative: CB, drug combination
taxane derivative: IT, drug interaction
taxane derivative: PD, pharmacology
gabapentin
amitriptyline
tramadol
carbamazepine
fentanyl: TD, transdermal drug administration
neuroprotective agent: AE, adverse drug reaction
neuroprotective agent: CT, clinical trial
neuroprotective agent: DT, drug therapy
neuroprotective agent: PD, pharmacology
corticotropin derivative: CT, clinical trial
corticotropin derivative: DT, drug therapy
corticotropin derivative: PD, pharmacology
org 2776: CT, clinical trial
org 2776: DT, drug therapy
org 2776: PD, pharmacology
thiol derivative: CT, clinical trial
thiol derivative: DT, drug therapy
thiol derivative: PD, pharmacology
glutathione: CT, clinical trial
glutathione: DT, drug therapy
glutathione: PD, pharmacology
amifostine: CT, clinical trial
amifostine: DT, drug therapy
amifostine: PD, pharmacology
dimesna: CT, clinical trial
dimesna: DT, drug therapy
dimesna: PD, pharmacology
alpha tocopherol: CT, clinical trial
alpha tocopherol: DT, drug therapy
alpha tocopherol: PD, pharmacology
growth factor: CT, clinical trial
growth factor: DT, drug therapy
growth factor: PD, pharmacology
neurotrophin 3
ciliary neurotrophic factor
somatomedin C: CT, clinical trial
somatomedin C: DT, drug therapy
glycoprotein gp 130
unclassified drug
wr 2711

CAS REGISTRY NO.: (vincristine) 57-22-7; (cisplatin) 15663-27-1, 26035-31-4,

96081-74-2; (oxaliplatin) 61825-94-3; (paclitaxel) 33069-62-4; (docetaxel) 114977-28-5; (bortezomib) 179324-69-7, 197730-97-5; (carboplatin) 41575-94-4; (gabapentin) 60142-96-3; (amitriptyline) 50-48-6, 549-18-8; (tramadol) 27203-92-5, 36282-47-0; (carbamazepine) 298-46-4, 8047-84-5; (fentanyl) 437-38-7; (thiol derivative) 13940-21-1; (glutathione) 70-18-8; (amifostine) 20537-88-6; **(dimesna) 16208-51-8, 45127-11-5**; (alpha tocopherol) 1406-18-4, 1406-70-8, 52225-20-4, 58-95-7, 59-02-9; (somatomedin C) 67763-96-6

CHEMICAL NAME: Org 2776; Wr 2711; **Bnp 7787**
COMPANY NAME: Cephalon (United States); Amrad (Australia)

L97 ANSWER 76 OF 90 EMBASE

ACCESSION NUMBER: 2004341830 EMBASE
TITLE: **BNP-7787.**
AUTHOR: Mealy N.E.
CORPORATE SOURCE: N.E. Mealy, Prous Science, P.O. Box 540, 08080 Barcelona, Spain
SOURCE: Drugs of the Future, (2004) Vol. 29, No. 6, pp. 629. .
ISSN: 0377-8282 CODEN: DRFUD4
COUNTRY: Spain
DOCUMENT TYPE: Journal; Note
FILE SEGMENT: 016 Cancer
028 Urology and Nephrology
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
ENTRY DATE: Entered STN: 2 Sep 2004
Last Updated on STN: 2 Sep 2004
CONTROLLED TERM: Medical Descriptors:
*neurotoxicity: DT, drug therapy
*neurotoxicity: PC, prevention
*neurotoxicity: SI, side effect
*nephrotoxicity: DT, drug therapy
*nephrotoxicity: PC, prevention
*nephrotoxicity: SI, side effect
solid tumor: DT, drug therapy
chemoprophylaxis
drug efficacy
breast cancer: DT, drug therapy
lung non small cell cancer: DT, drug therapy
human
clinical trial
phase 1 clinical trial
phase 3 clinical trial
note
Drug Descriptors:
***protective agent: CT, clinical trial**
***protective agent: DT, drug therapy**
*tavocept
taxane derivative: AE, adverse drug reaction
taxane derivative: CT, clinical trial
taxane derivative: DT, drug therapy
platinum derivative: AE, adverse drug reaction
platinum derivative: CT, clinical trial
platinum derivative: DT, drug therapy

cisplatin: AE, adverse drug reaction
cisplatin: CT, clinical trial
cisplatin: DT, drug therapy
paclitaxel: AE, adverse drug reaction
paclitaxel: CT, clinical trial
paclitaxel: DT, drug therapy
unclassified drug
bnp 7787

CAS REGISTRY NO.: (cisplatin) 15663-27-1, 26035-31-4, 96081-74-2; (paclitaxel) 33069-62-4
CHEMICAL NAME: (1) Tavocept; **(2) Bnp 7787**
COMPANY NAME: (2) Bionumerik

L97 ANSWER 77 OF 90 EMBASE

ACCESSION NUMBER: 2004292869 EMBASE
TITLE: Possible (enzymatic) routes and biological sites for metabolic reduction of BNP7787, a new protector against cisplatin-induced side-effects.
AUTHOR: Verschraagen M.; Boven E.; Torun E.; Hausheer F.H.; Bast A.; Van Der Vijgh W.J.F.
CORPORATE SOURCE: W.J.F. Van Der Vijgh, Department of Medical Oncology, Vrije Universiteit Medical Center, De Boelelaan 1117, 1007MB Amsterdam, Netherlands. wjf.vandervijgh@vumc.nl
SOURCE: Biochemical Pharmacology, (1 Aug 2004) Vol. 68, No. 3, pp. 493-502. .
Refs: 22
ISSN: 0006-2952 CODEN: BCPCA6
PUBLISHER IDENT.: S 0006-2952(04)00237-0
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 028 Urology and Nephrology
030 Pharmacology
037 Drug Literature Index
052 Toxicology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 5 Aug 2004
Last Updated on STN: 5 Aug 2004

ABSTRACT: Disodium 2,2'-dithio-bis-ethane sulfonate (BNP7787) is under investigation as a potential new chemoprotector against cisplatin-induced nephrotoxicity. The selective protection of BNP7787 appears to arise from the preferential uptake of the drug in the kidneys, where BNP7787 would undergo intracellular conversion into mesna (2-mercapto ethane sulfonate), which in turn can prevent cisplatin induced toxicities. In the present study, we have investigated whether the reduction of BNP7787 into the reactive compound mesna is restricted to the kidney or whether it can also occur in other organs, cells and physiological compartments, including the cytosolic fraction of the renal cortex, plasma, red blood cells (RBCs), liver and small intestine from rats and several tumors (OVCAR-3, MRI-H-207 and WARD). We also determined whether the endogenous thiols glutathione (GSH) and cysteine and the enzyme systems glutaredoxin and thioredoxin, which are all present in the kidney, can be involved in the BNP7787 reduction. UV detection and micro-HPLC with dual electrochemical detection were used to analyze the various incubation mixtures. Our observations are that, in contrast to plasma, a very large reductive conversion of BNP7787 to mesna was measured in RBC lysate. Intact RBCs, however, did not take up BNP7787. Although BNP7787 could be reduced in cytosol of liver and several tumors, this reduction will not be relevant in vivo, since these tissues do not take up large amounts of BNP7787. Kidney cortex cytosol was, similar to the small intestine cytosol, able to substantially reduce

BNP7787 to mesna. The ability to reduce BNP7787 in the presence of the endogenous thiols GSH and cysteine, the glutaredoxin system as well as the thioredoxin system, could at least in part explain the high BNP7787 reductive activity of the kidney cortex cytosol. In conclusion, the high reduction of BNP7787 into mesna in the kidney as well as our earlier observation that the distribution of BNP7787 and mesna was mainly restricted to rat kidney are strong arguments in favor of selective protection of the kidney by BNP7787.

CONTROLLED TERM: Medical Descriptors:
*nephrotoxicity: PC, prevention
*drug induced disease: PC, prevention
*protection
drug effect
drug potency
drug mechanism
cytosolic fraction
radiation detection
kidney cortex
erythrocyte
blood
liver
small intestine
in vivo study
enzyme activity
chemoprophylaxis
drug cytotoxicity: PC, prevention
nonhuman
rat
animal experiment
animal model
controlled study
animal tissue
article
priority journal
Drug Descriptors:
*disodium 2,2' dithiobis ethane sulfonate: PK, pharmacokinetics
*disodium 2,2' dithiobis ethane sulfonate: PD, pharmacology
***protective agent: PK, pharmacokinetics**
***protective agent: PD, pharmacology**
cisplatin: TO, drug toxicity
glutathione: EC, endogenous compound
glutaredoxin: EC, endogenous compound
thioredoxin: EC, endogenous compound
cysteine: EC, endogenous compound
unclassified drug
bnp 7787

CAS REGISTRY NO.: (cisplatin) 15663-27-1, 26035-31-4, 96081-74-2; (glutathione) 70-18-8; (glutaredoxin) 157514-02-8; (thioredoxin) 52500-60-4; (cysteine) 4371-52-2, 52-89-1, 52-90-4

CHEMICAL NAME: **(1) Bnp 7787**

COMPANY NAME: (1) Bionumerik

L97 ANSWER 78 OF 90 EMBASE

ACCESSION NUMBER: 2003264230 EMBASE

TITLE: Pharmacokinetics of BNP7787 and its metabolite mesna in plasma and ascites: A case report.

AUTHOR: Verschraagen M.; Boven E.; Zegers I.; Hausheer F.H.; Van Der Vijgh W.J.F.
CORPORATE SOURCE: M. Verschraagen, Department of Medical Oncology, Vrije Universiteit Medical Center, De Boelelaan 1117, 1007 MB Amsterdam, Netherlands. M.Verschraagen@vumc.nl
SOURCE: Cancer Chemotherapy and Pharmacology, (1 Jun 2003) Vol. 51, No. 6, pp. 525-529. .
Refs: 15
ISSN: 0344-5704 CODEN: CCPHDZ
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 24 Jul 2003
Last Updated on STN: 24 Jul 2003
ABSTRACT: Purpose: BNP7787 (2',2'-dithio-bis-ethane sulfonate sodium) is a novel protector against cisplatin-induced toxicities. The pharmacokinetics of BNP7787 and its metabolite mesna were investigated in plasma and ascites of a cancer patient. We also evaluated potential pharmacokinetic interactions between BNP7787 and cisplatin. Methods: BNP7787 and mesna were measured as mesna in deproteinized plasma and ascites using high-performance liquid chromatography with an electrochemical detector provided with a wall-jet gold electrode. Results: After the i.v. administration of 41 g/m(2) BNP7787, BNP7787 and mesna had a half-life of 1.5 and 3.4 h, respectively. The auc(∞) of mesna was approximately 8% of the AUC(∞) of BNP7787. Coadministration of cisplatin did not appear to influence the plasma concentration-time curves of BNP7787 and mesna. In ascites, approximately 0.02% of the BNP7787 dose was present as mesna, whereas approximately 4% of the dose was present as BNP7787 at the time of the maximum concentration. Conclusions: It can be concluded that the presence of ascites did not have a major impact on the pharmaco-kinetics of BNP7787 and coadministration of cisplatin did not influence the pharmacokinetics of BNP7787 and mesna.

CONTROLLED TERM: Medical Descriptors:
*plasma
*ascites: ET, etiology
cancer patient
high performance liquid chromatography
electrochemical detection
drug half life
area under the curve
drug metabolism
mean residence time
drug distribution
drug blood level
stomach cancer: DI, diagnosis
stomach cancer: DT, drug therapy
side effect: DT, drug therapy
side effect: SI, side effect
human
male
case report
adult
article
priority journal
Drug Descriptors:

***protective agent: CB, drug combination**
***protective agent: CR, drug concentration**
***protective agent: IT, drug interaction**
***protective agent: DT, drug therapy**
***protective agent: PK, pharmacokinetics**
***protective agent: IV, intravenous drug administration**
*2',2' dithiobisethanesulfonate sodium: CB, drug combination
*2',2' dithiobisethanesulfonate sodium: CR, drug concentration
*2',2' dithiobisethanesulfonate sodium: IT, drug interaction
*2',2' dithiobisethanesulfonate sodium: DT, drug therapy
*2',2' dithiobisethanesulfonate sodium: PK, pharmacokinetics
*2',2' dithiobisethanesulfonate sodium: IV, intravenous drug administration
*drug metabolite: CR, drug concentration
*drug metabolite: PK, pharmacokinetics
*mesna: CR, drug concentration
*mesna: PK, pharmacokinetics
cisplatin: AE, adverse drug reaction
cisplatin: CB, drug combination
cisplatin: IT, drug interaction
cisplatin: DT, drug therapy
cisplatin: IV, intravenous drug administration
unclassified drug
bnp 7787

CAS REGISTRY NO.: (mesna) 19767-45-4, 3375-50-6; (cisplatin) 15663-27-1, 26035-31-4, 96081-74-2
CHEMICAL NAME: **(1) Bnp 7787**
COMPANY NAME: (1) Bionumerik (United States)

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ACCESSION NUMBER: 2003264226 EMBASE
TITLE: The chemical reactivity of BNP7787 and its metabolite mesna with the cytostatic agent cisplatin: Comparison with the nucleophiles thiosulfate, DDTC, glutathione and its disulfide GSSG.
AUTHOR: Verschraagen M.; Kedde M.A.; Hausheer F.H.; Van Der Vijgh W.J.F.
CORPORATE SOURCE: M. Verschraagen, Department of Medical Oncology, Vrije Universiteit Medical Center, De Boelelaan 1117, 1007 MB Amsterdam, Netherlands. M.Verschraagen@vumc.nl
SOURCE: Cancer Chemotherapy and Pharmacology, (1 Jun 2003) Vol. 51, No. 6, pp. 499-504. .
Refs: 26
ISSN: 0344-5704 CODEN: CCPHDZ
COUNTRY: Germany
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
029 Clinical Biochemistry
037 Drug Literature Index
052 Toxicology
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 24 Jul 2003
Last Updated on STN: 24 Jul 2003

ABSTRACT: Purpose: BNP7787 is a new chemoprotective agent presently under clinical investigation to protect against cisplatin-induced toxicities, especially nephrotoxicity and neurotoxicity. In the kidneys BNP7787 is postulated to undergo selective conversion into mesna, which can locally detoxify cisplatin. The reactivity of cisplatin with this new chemoprotective agent and with its metabolite mesna was investigated at clinically observed plasma concentrations and compared with the nucleophiles thiosulfate (TS) and DDTC, and with the endogenous compounds glutathione (GSH) and oxidized glutathione (GSSG). Methods: Reaction kinetics experiments were performed at 37°C and pH 7.4 in the presence of a high chloride concentration (0.15 M). The degradation of cisplatin was measured over time using HPLC with off-line flameless atomic absorption spectrophotometry. Results: The degradation half-lives of cisplatin (13.5 μM) with 17.2 mM BNP7787, 340 μM mesna and 17.2 mM mesna were 124 min, about 790 min and 73 min, respectively. Cisplatin reacted at least 9.5 times more slowly with 17.2 mM BNP7787 and 5.5 times more slowly with 17.2 mM mesna than with 17.2 mM of the modulating agents DDTC or TS (i.e. half-lives 11 and 13 min, respectively). The half-lives of cisplatin with 17.2 mM GSH and GSSG (i.e. 122 and 115 min, respectively) were comparable with the half-life obtained with BNP7787. The thiol mesna was shown to be a stronger nucleophile than its corresponding disulfide BNP7787. Conclusions: The much slower relative reactivity of BNP7787, the short residence of BNP7787 (approximately 2 h) and the much lower concentration of mesna in the circulation following BNP7787 administration precludes chemical inactivation of cisplatin in the circulation, and thus the antitumor activity of cisplatin is maintained.

CONTROLLED TERM: Medical Descriptors:
*chemoreactivity
chemical reaction kinetics
drug degradation
high performance liquid chromatography
atomic absorption spectrometry
half life time
drug inactivation
antineoplastic activity
drug structure
chemical structure
nephrotoxicity: ET, etiology
neurotoxicity: ET, etiology
article
priority journal
Drug Descriptors:
***protective agent: AN, drug analysis**
***protective agent: CB, drug combination**
***protective agent: CM, drug comparison**
***bnp 7787: AN, drug analysis**
***bnp 7787: CB, drug combination**
***bnp 7787: CM, drug comparison**
*mesna: AN, drug analysis
*mesna: CB, drug combination
*mesna: CM, drug comparison
*drug metabolite: AN, drug analysis
*drug metabolite: CB, drug combination
*drug metabolite: CM, drug comparison
*cisplatin: AN, drug analysis
*cisplatin: CB, drug combination
*cisplatin: TO, drug toxicity
*thiol derivative: AN, drug analysis
*thiol derivative: CB, drug combination
*thiol derivative: CM, drug comparison

thiosulfate: AN, drug analysis
thiosulfate: CB, drug combination
thiosulfate: CM, drug comparison
diethyldithiocarbamic acid: AN, drug analysis
diethyldithiocarbamic acid: CB, drug combination
diethyldithiocarbamic acid: CM, drug comparison
glutathione
glutathione disulfide
unclassified drug

CAS REGISTRY NO.: (mesna) 19767-45-4, 3375-50-6; (cisplatin) 15663-27-1, 26035-31-4, 96081-74-2; (thiol derivative) 13940-21-1; (thiosulfate) 14383-50-7; (diethyldithiocarbamic acid) 147-84-2, 148-18-5, 3699-30-7, 392-74-5; (glutathione) 70-18-8; (glutathione disulfide) 27025-41-8

CHEMICAL NAME: **(1) Bnp 7787**

COMPANY NAME: (1) Bionumerik (United States); Sigma (United States); Brocacef (Netherlands)

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ACCESSION NUMBER: 2003320254 EMBASE

TITLE: Pharmacokinetics and preliminary clinical data of the novel chemoprotectant BNP7787 and cisplatin and their metabolites.

AUTHOR: Verschraagen M.; Boven E.; Ruijter R.; Van Der Born K.; Berkhof J.; Hausheer F.H.; Van Der Vijgh W.J.F.

CORPORATE SOURCE: M. Verschraagen, Vrije Universiteit Medical Center, Department of Medical Oncology, KRIGO, De Boelelaan 1117, 1007 MB Amsterdam, Netherlands. M.Verschraagen@vumc.nl

SOURCE: Clinical Pharmacology and Therapeutics, (1 Aug 2003) Vol. 74, No. 2, pp. 157-169. .
Refs: 32
ISSN: 0009-9236 CODEN: CLPTAT

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 4 Sep 2003
Last Updated on STN: 4 Sep 2003

ABSTRACT: Introduction: BNP7787 (disodium 2,2'-dithio-bis-ethane sulfonate) is currently undergoing development as a chemoprotective agent to prevent common and serious cisplatin-induced side effects. In the kidneys, intestine, and liver, BNP7787 is believed to undergo intracellular conversion into 2-mercaptoethane sulfonate (mesna), which can locally inactivate toxic platinum species. Methods and Objectives: In a phase I trial, 25 patients with advanced solid tumors received a 1-hour intravenous infusion of 75 mg/m(2) cisplatin immediately preceded by a 15-minute intravenous infusion of BNP7787 every 3 weeks. For pharmacokinetic investigation of BNP7787 and mesna and a possible mutual pharmacokinetic interaction between BNP7787 and cisplatin, cisplatin and BNP7787 were also administered as single agents in 14 of 25 patients. The dose of BNP7787 was escalated from 4.1 to 41 g/m(2). Patients were also monitored for tumor response and possible side effects from BNP7787. Results: The maximum plasma concentration of mesna was reached approximately 1.7 hours after the start of the BNP7787 infusion. The maximum plasma concentration and area under the curve to infinity (AUC∞) of BNP7787 and mesna increased linearly with the dose. The mean volume of distribution of BNP7787 (±SD) was approximately 0.26 ± 0.08 L/kg. The mean normalized AUC∞ of mesna

was only approximately 8% of the normalized AUC∞ of BNP7787. The pharmacokinetic profile of mesna was unaffected by cisplatin and its metabolites. None of the dose levels of BNP7787 (4.1-41 g/m(2)) administered appeared to influence the pharmacokinetic profile of total platinum, unbound platinum, or monohydrated cisplatin. The observed effects regarding a possible mutual interaction between BNP7787 and intact cisplatin were minor, and none were statistically significant at BNP7787 dose levels of 18.4 to 41 g/m(2). The confidence intervals for the pharmacokinetic parameters of BNP7787 and intact cisplatin, however, were relatively broad. Overall, BNP7787 was well tolerated at all dose levels (4.1-41.0 g/m(2)). The most frequently reported event related to BNP7787 was local intravenous site discomfort; the majority of events were mild (grade 1). Side effects of BNP7787 at the highest dose level of 41 g/m(2) were more prominent and included nausea and vomiting, as well as a warm feeling or flushing (grade 2 or lower). Partial tumor responses and stable disease were measured in 12 of 25 patients. Conclusion: BNP7787 was relatively nontoxic at doses up to 41 g/m(2). The combination of BNP7787 with cisplatin did not alter the pharmacokinetic profiles of mesna or the cisplatin metabolites. At the higher dose levels of BNP7787 (18.4 to 41 g/m(2)), there appeared to be no mutual interaction between BNP7787 and intact cisplatin, which needs to be confirmed in a larger number of patients. The absence of a mutual interaction between BNP7787 and intact cisplatin is consistent with the observation that several patients had objective tumor responses with BNP7787 and cisplatin administration.

CONTROLLED TERM: Medical Descriptors:
drug blood level
drug distribution
area under the curve
drug tolerability
nausea: SI, side effect
vomiting: SI, side effect
flushing
side effect: SI, side effect
human
male
female
clinical article
clinical trial
phase 1 clinical trial
controlled study
adult
article
priority journal
Drug Descriptors:
*disodium 2,2' dithiobisethanesulfonate: CT, clinical trial
*disodium 2,2' dithiobisethanesulfonate: DV, drug development
*disodium 2,2' dithiobisethanesulfonate: DO, drug dose
*disodium 2,2' dithiobisethanesulfonate: IT, drug interaction
*disodium 2,2' dithiobisethanesulfonate: PK, pharmacokinetics
*disodium 2,2' dithiobisethanesulfonate: PD, pharmacology
*disodium 2,2' dithiobisethanesulfonate: IV, intravenous drug administration
***protective agent: CT, clinical trial**
***protective agent: DV, drug development**
***protective agent: DO, drug dose**
***protective agent: IT, drug interaction**
***protective agent: PK, pharmacokinetics**

***protective agent: PD, pharmacology**
***protective agent: IV, intravenous drug administration**
*cisplatin: AE, adverse drug reaction
*cisplatin: CT, clinical trial
*cisplatin: IT, drug interaction
*cisplatin: IV, intravenous drug administration
mesna: CR, drug concentration
mesna: PK, pharmacokinetics
mesna: PD, pharmacology
platinum: PK, pharmacokinetics
ondansetron
dexamethasone
unclassified drug
bnp 7787

CAS REGISTRY NO.: (cisplatin) 15663-27-1, 26035-31-4, 96081-74-2; (mesna) 19767-45-4, 3375-50-6; (platinum) 7440-06-4; (ondansetron) 103639-04-9, 116002-70-1, 99614-01-4; (dexamethasone) 50-02-2
CHEMICAL NAME: **(1) Bnp 7787**; (2) Platosin
COMPANY NAME: (1) Bionumerik (United States); (2) Pharmachemie (Netherlands)

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ACCESSION NUMBER: 2003350292 EMBASE
TITLE: New approaches to drug discovery and development: A mechanism-based approach to pharmaceutical research and its application to BNP7787, a novel chemoprotective agent.
AUTHOR: Hausheer F.H.; Kochat H.; Parker A.R.; Ding D.; Yao S.; Hamilton S.E.; Petluru P.N.; Leverett B.D.; Bain S.H.; Saxe J.D.
CORPORATE SOURCE: F.H. Hausheer, BioNumerik Pharmaceuticals, Inc., 8122 Datapoint Drive, San Antonio, TX 78229, United States. fred.hausheer@bnpi.com
SOURCE: Cancer Chemotherapy and Pharmacology, Supplement, (2003) Vol. 52, No. 1, pp. S3-S15. .
Refs: 27
ISSN: 0943-9404 CODEN: CCHSET
COUNTRY: Germany
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 016 Cancer
027 Biophysics, Bioengineering and Medical Instrumentation
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 18 Sep 2003
Last Updated on STN: 18 Sep 2003

ABSTRACT: Any approach applied to drug discovery and development by the medical community and pharmaceutical industry has a direct impact on the future availability of improved, novel, and curative therapies for patients with cancer. By definition, drug discovery is a complex learning process whereby research efforts are directed toward uncovering and assimilating new knowledge to create and develop a drug for the purpose of providing benefit to a defined patient population. Accordingly, a highly desirable technology or approach to drug discovery should facilitate both effective learning and the application of newly discovered observations that can be exploited for therapeutic benefit.

However, some believe that drug discovery is largely accomplished by serendipity and therefore appropriately addressed by screening a large number of compounds. Clearly, this approach has not generated an abundance of new drugs for cancer patients and suggests that a tangibly different approach in drug discovery is warranted. We employ an alternative approach to drug discovery, which is based on the elucidation and exploitation of biological, pharmacological, and biochemical mechanisms that have not been previously recognized or fully understood. Mechanism-based drug discovery involves the combined application of physics-based computer simulations and laboratory experimentation. There is increasing evidence that agreement between simulations based on the laws of physics and experimental observations results in a higher probability that such observations are more accurate and better understood as compared with either approach used alone. Physics-based computer simulation applied to drug discovery is now considered by experts in the field to be one of the ultimate methodologies for drug discovery. However, the ability to perform truly comprehensive physics-based molecular simulations remains limited by several factors, including the enormous computer-processing power that is required to perform the formidable mathematical operations and data processing (e.g. memory bandwidth, data storage and retrieval). Another major consideration is the development of software that can generate an appropriate and increasingly complex physical representation of the atomic arrangements of biological systems. During the past 17 years, we have made tremendous progress in addressing some of these obstacles by developing and optimizing physics-based computer programs for the purpose of obtaining increasingly accurate and precise information and by improving the speed of computation. To perform physics-based simulations that involve complex systems of biological and pharmaceutical interest, we have developed methods that enable us to exceed Moore's law. This has been accomplished by parallel processing as well as other methods that have enabled us to study more complex and relevant molecular systems of interest. This paper provides an overview of our approach to drug discovery and describes a novel drug, currently in clinical development, which has directly resulted from the application of this approach.

CONTROLLED TERM: Medical Descriptors:
drug mechanism
drug research
drug screening
drug industry
physics
computer simulation
experiment
computer program
diarrhea: SI, side effect
headache: SI, side effect
pain: SI, side effect
nausea: SI, side effect
hypotension: SI, side effect
lung non small cell cancer: DT, drug therapy
neurotoxicity: SI, side effect
nephrotoxicity: DT, drug therapy
nephrotoxicity: PC, prevention
nephrotoxicity: SI, side effect
ototoxicity: SI, side effect
bone marrow toxicity: SI, side effect
human
nonhuman
clinical trial
phase 1 clinical trial
phase 3 clinical trial

conference paper
priority journal
Drug Descriptors:
*disodium 2,2' dithiobisethanesulfonate: AE, adverse drug reaction
*disodium 2,2' dithiobisethanesulfonate: CT, clinical trial
*disodium 2,2' dithiobisethanesulfonate: AN, drug analysis
*disodium 2,2' dithiobisethanesulfonate: CB, drug combination
*disodium 2,2' dithiobisethanesulfonate: DV, drug development
*disodium 2,2' dithiobisethanesulfonate: DO, drug dose
*disodium 2,2' dithiobisethanesulfonate: DT, drug therapy
*disodium 2,2' dithiobisethanesulfonate: TO, drug toxicity
*disodium 2,2' dithiobisethanesulfonate: PK, pharmacokinetics
*disodium 2,2' dithiobisethanesulfonate: PD, pharmacology
*disodium 2,2' dithiobisethanesulfonate: IV, intravenous drug administration
*antineoplastic agent: AE, adverse drug reaction
*antineoplastic agent: CT, clinical trial
*antineoplastic agent: AN, drug analysis
*antineoplastic agent: CB, drug combination
*antineoplastic agent: DV, drug development
*antineoplastic agent: DO, drug dose
*antineoplastic agent: DT, drug therapy
*antineoplastic agent: TO, drug toxicity
*antineoplastic agent: PK, pharmacokinetics
*antineoplastic agent: PD, pharmacology
*antineoplastic agent: IV, intravenous drug administration
platinum derivative: AE, adverse drug reaction
platinum derivative: CT, clinical trial
platinum derivative: CB, drug combination
platinum derivative: DT, drug therapy
taxane derivative: AE, adverse drug reaction
taxane derivative: CT, clinical trial
taxane derivative: CB, drug combination
taxane derivative: DT, drug therapy
cisplatin: AE, adverse drug reaction
cisplatin: CT, clinical trial
cisplatin: CB, drug combination
cisplatin: DT, drug therapy
paclitaxel: AE, adverse drug reaction
paclitaxel: CT, clinical trial
paclitaxel: CB, drug combination
paclitaxel: DT, drug therapy
mesna: AE, adverse drug reaction
mesna: DO, drug dose
mesna: TO, drug toxicity
mesna: PK, pharmacokinetics
mesna: IV, intravenous drug administration
sodium chloride: DT, drug therapy
amifostine: PK, pharmacokinetics
amifostine: IV, intravenous drug administration
unclassified drug
bnp 7787
tavocept

CAS REGISTRY NO.: (cisplatin) 15663-27-1, 26035-31-4, 96081-74-2; (paclitaxel) 33069-62-4; (mesna) 19767-45-4, 3375-50-6; (sodium chloride) 7647-14-5; (amifostine) 20537-88-6

CHEMICAL NAME: **Bnp 7787**; Tavocept

L97 ANSWER 82 OF 90 EMBASE

ACCESSION NUMBER: 2002389867 EMBASE
TITLE: Current status and future prospects for the treatment of chemotherapy-induced peripheral neurotoxicity.
AUTHOR: Cavaletti G.; Zanna C.
CORPORATE SOURCE: G. Cavaletti, Clinica Neurologia - A.O.S. Gerardo, v. Donizetti 106, 20052 Monza (MI), Italy. guido.cavaletti@unimib.it
SOURCE: European Journal of Cancer, (2002) Vol. 38, No. 14, pp. 1832-1837. .
Refs: 54
ISSN: 0959-8049 CODEN: EJCAEL
PUBLISHER IDENT.: S 0959-8049(02)00229-0
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 008 Neurology and Neurosurgery
016 Cancer
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
ENTRY DATE: Entered STN: 21 Nov 2002
Last Updated on STN: 21 Nov 2002
CONTROLLED TERM: Medical Descriptors:
*cancer chemotherapy
*neurotoxicity: DT, drug therapy
*neurotoxicity: SI, side effect
neuroprotection
drug effect
paresthesia
pain
human
nonhuman
clinical trial
review
priority journal
Drug Descriptors:
*antineoplastic agent: AE, adverse drug reaction
*antineoplastic agent: TO, drug toxicity
cisplatin: AE, adverse drug reaction
cisplatin: TO, drug toxicity
paclitaxel: AE, adverse drug reaction
paclitaxel: TO, drug toxicity
2 amino 4 (methylsulfonyl)butyrylglutamylhistidylphenylalanyl dextro lysylphenylalanine: CT, clinical trial
2 amino 4 (methylsulfonyl)butyrylglutamylhistidylphenylalanyl dextro lysylphenylalanine: DO, drug dose
2 amino 4 (methylsulfonyl)butyrylglutamylhistidylphenylalanyl dextro lysylphenylalanine: DT, drug therapy
amifostine: CT, clinical trial
alkylating agent: AE, adverse drug reaction
carboplatin: AE, adverse drug reaction
glutathione: CT, clinical trial
leukemia inhibitory factor: CT, clinical trial
leukemia inhibitory factor: PD, pharmacology
somatomedin C: CT, clinical trial
somatomedin C: PD, pharmacology
vincristine: TO, drug toxicity

neurotrophin 3: PD, pharmacology
nerve growth factor: CB, drug combination
nerve growth factor: PD, pharmacology
levacecarnine: CT, clinical trial
levacecarnine: CB, drug combination
levacecarnine: PD, pharmacology
glutamic acid: CT, clinical trial
glutamine: CT, clinical trial
leteprinim: CT, clinical trial
lithium: CT, clinical trial
alpha tocopherol: CT, clinical trial
xaliproden: CT, clinical trial
glial cell line derived neurotrophic factor: CT, clinical trial
ciliary neurotrophic factor: CT, clinical trial
Vinca alkaloid: AE, adverse drug reaction
bnp 7787
wr 2771

CAS REGISTRY NO.: (cisplatin) 15663-27-1, 26035-31-4, 96081-74-2; (paclitaxel) 33069-62-4; (2 amino 4 (methylsulfonyl)butyrylglutamylhistidylphenylalanyl dextro lysylphenylalanine) 50913-82-1; (amifostine) 20537-88-6; (carboplatin) 41575-94-4; (glutathione) 70-18-8; (somatomedin C) 67763-96-6; (vincristine) 57-22-7; (nerve growth factor) 9061-61-4; (levacecarnine) 3040-38-8, 5080-50-2; (glutamic acid) 11070-68-1, 138-15-8, 56-86-0, 6899-05-4; (glutamine) 56-85-9, 6899-04-3; (leteprinim) 138117-50-7, 192564-13-9; (lithium) 7439-93-2; (alpha tocopherol) 1406-18-4, 1406-70-8, 52225-20-4, 58-95-7, 59-02-9; (xaliproden) 90494-79-4

CHEMICAL NAME: Ait 082; **Bnp 7787**; Wr 2771; Org 2766

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ACCESSION NUMBER: 2000197998 EMBASE
TITLE: Clinical perspectives on platinum resistance.
AUTHOR: Giaccone G.
CORPORATE SOURCE: G. Giaccone, Department of Medical Oncology, Academic Hospital, Vrije Universiteit, 1117 De Boelelaan, HV1081 Amsterdam, Netherlands
SOURCE: Drugs, (2000) Vol. 59, No. SUPPL. 4, pp. 9-17. .
Refs: 56
ISSN: 0012-6667 CODEN: DRUGAY
COUNTRY: New Zealand
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 037 Drug Literature Index
038 Adverse Reactions Titles
016 Cancer
030 Pharmacology
022 Human Genetics
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 30 Jun 2000
Last Updated on STN: 30 Jun 2000

ABSTRACT: The platinum compounds cisplatin and carboplatin are widely used in the treatment of a number of solid malignancies. Although some platinum-sensitive tumours may be cured by combination chemotherapy (e.g. testicular cancer), most will relapse and subsequently prove resistant to platinum compounds. The mechanisms of platinum resistance in patients are still poorly understood. Clearly, when a tumour relapses a long time after

successful first-line treatment, there is a high chance that it will still be sensitive to platinum compounds. A number of studies have attempted to assess the role of drug transport, the glutathione system, DNA repair and apoptosis genes in the development of resistance in tumours, but no conclusive evidence is available. Approaches to increasing the potency of platinum therapy (to overcome resistance) have been devised and some have proved to be effective; in particular, intraperitoneal administration of cisplatin has shown superiority over intravenous administration in selected patients with ovarian cancer. The development of drugs and techniques to reduce the adverse effects of platinum chemotherapy has greatly improved their administration. Investigations attempting to modulate platinum activity and toxicity have also been performed. Further investigation of in vivo resistance mechanisms should be valuable in allowing prediction of clinical response to chemotherapy and may identify new treatments with the potential to improve outcomes for patients with a variety of platinum-resistant tumour types.

CONTROLLED TERM: Medical Descriptors:
*cancer: DT, drug therapy
*cancer: DR, drug resistance
human
drug transport
glutathione metabolism
DNA repair
apoptosis
tumor suppressor gene
drug potency
cancer combination chemotherapy
lung small cell cancer: DT, drug therapy
lung small cell cancer: DR, drug resistance
lung small cell cancer: RT, radiotherapy
ovary cancer: DT, drug therapy
ovary cancer: DR, drug resistance
testis cancer: DT, drug therapy
testis cancer: DR, drug resistance
head cancer: DT, drug therapy
head cancer: DR, drug resistance
neck cancer: DT, drug therapy
neck cancer: DR, drug resistance
lung non small cell cancer: DT, drug therapy
lung non small cell cancer: DR, drug resistance
vomiting: SI, side effect
vomiting: DT, drug therapy
nephrotoxicity: SI, side effect
nephrotoxicity: DT, drug therapy
neurotoxicity: SI, side effect
neurotoxicity: DT, drug therapy
drug formulation
review
Drug Descriptors:
*cisplatin: DT, drug therapy
*cisplatin: PD, pharmacology
*cisplatin: CB, drug combination
*cisplatin: IP, intraperitoneal drug administration
*cisplatin: IV, intravenous drug administration
*cisplatin: AD, drug administration
*cisplatin: DO, drug dose
*cisplatin: AE, adverse drug reaction
*cisplatin: PR, pharmaceutics
*carboplatin: DT, drug therapy
*carboplatin: PD, pharmacology

*carboplatin: CB, drug combination
*carboplatin: IP, intraperitoneal drug administration
*carboplatin: IV, intravenous drug administration
*carboplatin: AD, drug administration
*carboplatin: DO, drug dose
*carboplatin: AE, adverse drug reaction
*carboplatin: PR, pharmaceutics
etoposide: DT, drug therapy
etoposide: CB, drug combination
taxol: DT, drug therapy
taxol: CB, drug combination
bleomycin: DT, drug therapy
bleomycin: CB, drug combination
fluorouracil: DT, drug therapy
fluorouracil: CB, drug combination
glutathione: EC, endogenous compound
DNA: EC, endogenous compound
serotonin antagonist: DT, drug therapy
bnp 7787: DT, drug therapy
amifostine: DT, drug therapy
oxaliplatin: DV, drug development
amminedichloro(2 methylpyridine)platinum: DV, drug development
cyclosporin: DT, drug therapy
cyclosporin: CB, drug combination
dipyridamole: DT, drug therapy
dipyridamole: CB, drug combination
amphotericin B: DT, drug therapy
amphotericin B: CB, drug combination
trifluoperazine: DT, drug therapy
trifluoperazine: CB, drug combination
buthionine sulfoximine: DT, drug therapy
buthionine sulfoximine: CB, drug combination
aphidicolin: DT, drug therapy
aphidicolin: CB, drug combination
novobiocin: DT, drug therapy
novobiocin: CB, drug combination
cytarabine: DT, drug therapy
cytarabine: CB, drug combination
hydroxyurea: DT, drug therapy
hydroxyurea: CB, drug combination
gemcitabine: DT, drug therapy
gemcitabine: CB, drug combination
DNA polymerase: EC, endogenous compound
DNA topoisomerase (ATP hydrolysing): EC, endogenous compound
unclassified drug

CAS REGISTRY NO.: (cisplatin) 15663-27-1, 26035-31-4, 96081-74-2; (carboplatin) 41575-94-4; (etoposide) 33419-42-0; (taxol) 33069-62-4; (bleomycin) 11056-06-7; (fluorouracil) 51-21-8; (glutathione) 70-18-8; (DNA) 9007-49-2; (amifostine) 20537-88-6; (oxaliplatin) 61825-94-3; (amminedichloro(2 methylpyridine)platinum) 181630-15-9; (cyclosporin) 79217-60-0; (dipyridamole) 58-32-2; (amphotericin B) 1397-89-3, 30652-87-0; (trifluoperazine) 117-89-5, 440-17-5; (buthionine sulfoximine) 5072-26-4; (aphidicolin) 38966-21-1; (novobiocin) 1476-53-5, 303-81-1, 39301-00-3, 4309-70-0; (cytarabine) 147-94-4, 69-74-9; (hydroxyurea) 127-07-1; (gemcitabine) 103882-84-4; (DNA polymerase) 37217-33-7

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ACCESSION NUMBER: 1999315273 EMBASE
TITLE: A novel PARP inhibitor, ion channel modulation and AD therapies.
AUTHOR: Worker C.
CORPORATE SOURCE: C. Worker, Current Drugs Ltd, Middlesex House, 34-42 Cleveland Street, London W1P 6LB, United Kingdom. charlotte@cursci.co.uk
SOURCE: IDrugs, (1999) Vol. 2, No. 9, pp. 859-860. .
ISSN: 1369-7056 CODEN: IDRUFN
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 037 Drug Literature Index
030 Pharmacology
038 Adverse Reactions Titles
018 Cardiovascular Diseases and Cardiovascular Surgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 7 Oct 1999
Last Updated on STN: 7 Oct 1999

ABSTRACT: On the fourth and final day of the EPHAR congress, ion channel modulation was the topic for two symposia and plenary lectures. The potential of dual potassium and calcium channel blockers as antiarrhythmics was discussed, amongst other applications for ion channel modifiers. Several presentations were dedicated to the disclosure of a novel PARP inhibitor, BGP-15, developed at the University Medical School of Pecs in Hungary. This compound is emerging as a promising potential anti-ischemic and a chemoprotective agent. The treatment of Alzheimer's disease (AD) was the subject of further discussions; a detailed presentation was given by a psychiatrist from the US, describing the treatment regimens favored in her clinic, as well as a complete review of currently available and potentially new AD therapies.

CONTROLLED TERM: Medical Descriptors:
*Alzheimer disease: DT, drug therapy
nonhuman
mouse
animal model
drug efficacy
drug mechanism
ischemia: DT, drug therapy
antiarrhythmic activity
heart arrhythmia: DT, drug therapy
antineoplastic activity
cognitive defect: DT, drug therapy
drug antagonism
liver toxicity
nausea: SI, side effect
vomiting: SI, side effect
bleeding: SI, side effect
conference paper
Drug Descriptors:
*bgp 15: DT, drug therapy
*bgp 15: DV, drug development
*bgp 15: PD, pharmacology
*bgp 15: CB, drug combination
*bgp 15: CM, drug comparison
*nicotinamide adenine dinucleotide adenosine diphosphate

ribosyltransferase: EC, endogenous compound
*calcium channel blocking agent: DT, drug therapy
*calcium channel blocking agent: PD, pharmacology
*calcium channel blocking agent: CB, drug combination
*calcium channel blocking agent: IT, drug interaction
*potassium channel blocking agent: DT, drug therapy
*potassium channel blocking agent: PD, pharmacology
*potassium channel blocking agent: CB, drug combination
*potassium channel blocking agent: IT, drug interaction
reactive oxygen metabolite: EC, endogenous compound
nicotinamide adenine dinucleotide: EC, endogenous compound
grp 78: PD, pharmacology
amifostine: DT, drug therapy
amifostine: CB, drug combination
amifostine: PD, pharmacology
razoxane: DT, drug therapy
razoxane: CB, drug combination
razoxane: PD, pharmacology
bnp 7787: DT, drug therapy
bnp 7787: CB, drug combination
bnp 7787: PD, pharmacology
cytostatic agent: CB, drug combination
cytostatic agent: PD, pharmacology
doxorubicin: DT, drug therapy
doxorubicin: CB, drug combination
doxorubicin: PD, pharmacology
cisplatin: DT, drug therapy
cisplatin: CB, drug combination
cisplatin: PD, pharmacology
cisplatin: CM, drug comparison
antiarrhythmic agent: DT, drug therapy
antiarrhythmic agent: IT, drug interaction
antiarrhythmic agent: CB, drug combination
n (3,4 dimethoxyphenyl) n [3 [n [2 (3,4 dimethoxyphenyl)ethyl] n methylamino]propyl] 4 nitrobenzamide: PD, pharmacology
n (3,4 dimethoxyphenyl) n [3 [n [2 (3,4 dimethoxyphenyl)ethyl] n methylamino]propyl] 4 nitrobenzamide: DT, drug therapy
sb 237376: PD, pharmacology
sb 237376: DT, drug therapy
dofetilide: DT, drug therapy
dofetilide: CM, drug comparison
dofetilide: CB, drug combination
dofetilide: PD, pharmacology
tacrine: DT, drug therapy
tacrine: PD, pharmacology
tacrine: AE, adverse drug reaction
donepezil: DT, drug therapy
donepezil: PD, pharmacology
donepezil: AE, adverse drug reaction
alpha tocopherol: DT, drug therapy
flavonoid: DT, drug therapy
resveratol: DT, drug therapy
selegiline: DT, drug therapy
ubiquinone: DT, drug therapy
ginkgo biloba extract: DT, drug therapy
ginkgo biloba extract: AE, adverse drug reaction
conjugated estrogen: DT, drug therapy
conjugated estrogen: AE, adverse drug reaction

nonsteroid antiinflammatory agent: DT, drug therapy
nonsteroid antiinflammatory agent: AE, adverse drug reaction
memantine: DT, drug therapy
memantine: AE, adverse drug reaction
unindexed drug
dimesna

CAS REGISTRY NO.: (nicotinamide adenine dinucleotide adenosine diphosphate ribosyltransferase) 58319-92-9, 9055-67-8; (nicotinamide adenine dinucleotide) 53-84-9; (amifostine) 20537-88-6; (razoxane) 21416-67-1, 21416-87-5, 24584-09-6, 24613-06-7; (doxorubicin) 23214-92-8, 25316-40-9; (cisplatin) 15663-27-1, 26035-31-4, 96081-74-2; (dofetilide) 115256-11-6; (tacrine) 1684-40-8, 3198-41-2, 321-64-2; (donepezil) 120011-70-3, 120014-06-4, 142057-77-0; (alpha tocopherol) 1406-18-4, 1406-70-8, 52225-20-4, 58-95-7, 59-02-9; (selegiline) 14611-51-9, 14611-52-0, 2079-54-1, 2323-36-6; (ubiquinone) 1339-63-5; (memantine) 19982-08-2, 41100-52-1; (**dimesna**) **16208-51-8**, **45127-11-5**

CHEMICAL NAME: **(1) Dimesna**; (2) Brl 32872; (3) Sb 237376; Bgp 15; Grp 78; **Bnp 7787**; Premarin

COMPANY NAME: (1) Bionumerik pharmaceuticals; (3) Smith Kline Beecham; Imperial Cancer Research; United States Bioscience; Esai; Pfizer; Warner Lambert; Somerset

L97 ANSWER 85 OF 90 EMBASE

ACCESSION NUMBER: 2000060479 EMBASE

TITLE: Platinum neurotoxicity: Clinical profiles, experimental models and neuroprotective approaches.

AUTHOR: Screnci D.; McKeage M.J.

CORPORATE SOURCE: M.J. McKeage, Dept. of Pharmacol./Clin. Pharmacol., University of Auckland, Private Bag 92019, Auckland, New Zealand. m.mckeage@auckland.ac.nz

SOURCE: Journal of Inorganic Biochemistry, (1999) Vol. 77, No. 1-2, pp. 105-110. .
Refs: 76
ISSN: 0162-0134 CODEN: JIBIDJ

PUBLISHER IDENT.: S 0162-0134(99)00135-X

COUNTRY: United States

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 008 Neurology and Neurosurgery
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
052 Toxicology

LANGUAGE: English

SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 24 Feb 2000
Last Updated on STN: 24 Feb 2000

ABSTRACT: This paper reviews the neurotoxic side-effects associated with platinum drugs, experimental approaches to studying this toxicity and attempts to use neuroprotective agents in conjunction with platinum drugs. Platinum drugs differ in their neurotoxicity profiles in patients. The frequency, severity, mode of onset and reversibility of peripheral nerve toxicity varies between different platinum analogues. Animal models, primary cultures of dorsal root ganglia neurons and tumour cell-lines of neuronal origin are being used in attempts to identify potential treatments for platinum-induced neurotoxicity. To date, clinical trials have been hampered by the poor

tolerance of neuroprotective treatments and failure to achieve reversal of platinum drug neurotoxicity with thiols, neurotrophic factors or calcium channel blockers. (C) 2000 Elsevier Science Inc.

CONTROLLED TERM: Medical Descriptors:
*neurotoxicity: SI, side effect
peripheral neuropathy: SI, side effect
spinal ganglion
tumor cell culture
drug induced disease: SI, side effect
human
nonhuman
review
Drug Descriptors:
*platinum derivative: AE, adverse drug reaction
*platinum derivative: CB, drug combination
*platinum derivative: CM, drug comparison
*platinum derivative: TO, drug toxicity
*neuroprotective agent: AE, adverse drug reaction
*neuroprotective agent: CB, drug combination
*neuroprotective agent: CM, drug comparison
thiol derivative: AE, adverse drug reaction
thiol derivative: CB, drug combination
thiol derivative: CM, drug comparison
neurotrophic factor
calcium channel blocking agent: AE, adverse drug reaction
calcium channel blocking agent: CB, drug combination
oxaliplatin: AE, adverse drug reaction
tetraplatin: AE, adverse drug reaction
cisplatin: AE, adverse drug reaction
cisplatin: CB, drug combination
cisplatin: CM, drug comparison
sebriplatin: AE, adverse drug reaction
satraplatin: AE, adverse drug reaction
carboplatin: AE, adverse drug reaction
iproplatin: AE, adverse drug reaction
lobaplatin: AE, adverse drug reaction
zeniplatin: AE, adverse drug reaction
glutathione: CB, drug combination
bnp 7787
diethyldithiocarbamic acid: CB, drug combination
diethyldithiocarbamic acid: CM, drug comparison
cyclophosphamide: CB, drug combination
cyclophosphamide: CM, drug comparison
etoposide: CB, drug combination
etoposide: CM, drug comparison
amifostine: CB, drug combination
amifostine: CM, drug comparison
neurotrophin 3
2 amino 4 (methylsulfonyl)butyrylglutamylhistidylphenylalanyl dextro lysylphenylalanine
neurotrophin 4
nerve growth factor
brain derived neurotrophic factor
basic fibroblast growth factor
ciliary neurotrophic factor
nimodipine: CB, drug combination
taxol: CB, drug combination
unindexed drug

CAS REGISTRY NO.: (thiol derivative) 13940-21-1; (oxaliplatin) 61825-94-3;

(tetraplatin) 62816-98-2, 96392-95-9, 96392-96-0, 96392-97-1; (cisplatin) 15663-27-1, 26035-31-4, 96081-74-2; (sebriplatin) 110172-45-7; (satraplatin) 129580-63-8; (carboplatin) 41575-94-4; (iproplatin) 62928-11-4; (lobaplatin) 135558-11-1; (zeniplatin) 111490-36-9; (glutathione) 70-18-8; (diethyldithiocarbamic acid) 147-84-2, 148-18-5, 3699-30-7, 392-74-5; (cyclophosphamide) 50-18-0; (etoposide) 33419-42-0; (amifostine) 20537-88-6; (2 amino 4 (methylsulfonyl)butyrylglutamylhistidylphenylalanyl dextro lysylphenylalanine) 50913-82-1; (nerve growth factor) 9061-61-4; (basic fibroblast growth factor) 106096-93-9; (nimodipine) 66085-59-4; (taxol) 33069-62-4

CHEMICAL NAME: Ci 973; Jm 216; **Bnp 7787**; Org 2766

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ACCESSION NUMBER: 93040436 EMBASE
DOCUMENT NUMBER: 1993040436
TITLE: Prevention of singlet oxygen-induced DNA damage by lipoate.
AUTHOR: Devasagayam T.P.A.; Subramanian M.; Pradhan D.S.; Sies H.
CORPORATE SOURCE: Institut fur Physiologische Chemie I, Universitat Dusseldorf, Moorenstrasse 5,W-4000 Dusseldorf, Germany
SOURCE: Chemico-Biological Interactions, (1993) Vol. 86, No. 1, pp. 79-92. .
ISSN: 0009-2797 CODEN: CBINA8
COUNTRY: Ireland
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
005 General Pathology and Pathological Anatomy
029 Clinical Biochemistry
052 Toxicology
030 Pharmacology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 7 Mar 1993
Last Updated on STN: 7 Mar 1993

ABSTRACT: Among the several biologically and pharmacologically active sulfur compounds examined, only lipoic acid and dihydrolipoic acid provided protection to plasmid DNA against singlet molecular oxygen (1O2). 1O2 was generated in phosphate buffer by the thermal dissociation of the endoperoxide of 3,3'-(1,4-naphthylidene) dipropionate (NDPO2). The protecting effect of lipoic acid was time- and pH-dependent and significant protection was seen even at 50 μM. The antioxidant effect was adversely affected by temperature above 45°C. Superoxide dismutase and catalase marginally enhanced this effect. Metal chelation with EDTA decreased the protection by lipoate, indicating that metal ions may be involved. The protective effect was diminished when the disulfide was added after single-strand breaks were induced by 1O2. The formation of 8-oxoguanosine from guanosine upon exposure to NDPO2 was not altered by lipoate.

CONTROLLED TERM: Medical Descriptors:
*antioxidant activity
*dna damage
article
controlled study
dna strand breakage
escherichia coli
nonhuman
ph

prevention
priority journal
temperature
Drug Descriptors:
*antioxidant: PD, pharmacology
*plasmid dna
*singlet oxygen: TO, drug toxicity
*thioctic acid: PD, pharmacology
3,3' (1,4 naphthylidene)dipropionate: TO, drug toxicity
acetylcysteine: PD, pharmacology
amifostine: PD, pharmacology
captopril: PD, pharmacology
catalase: PD, pharmacology
cysteine: PD, pharmacology
cystine: PD, pharmacology
dihydrolipoate: PD, pharmacology
dimesna: PD, pharmacology
dithiothreitol: PD, pharmacology
edetic acid
glutathione: PD, pharmacology
glutathione disulfide: PD, pharmacology
mannitol: PD, pharmacology
mesna: PD, pharmacology
penicillamine: PD, pharmacology
superoxide dismutase: PD, pharmacology
thiol derivative: PD, pharmacology
thioneine: PD, pharmacology
tiopronin: PD, pharmacology
unclassified drug
CAS REGISTRY NO.: (thioctic acid) 1077-29-8, 1200-22-2, 2319-84-8, 62-46-4; (acetylcysteine) 616-91-1; (amifostine) 20537-88-6; (captopril) 62571-86-2; (catalase) 9001-05-2; (cysteine) 4371-52-2, 52-89-1, 52-90-4; (cystine) 24645-67-8, 56-89-3, 6020-39-9; (dihydrolipoate) 462-20-4; (**dimesna**) **16208-51-8**, **45127-11-5**; (dithiothreitol) 3483-12-3; (edetic acid) 150-43-6, 60-00-4; (glutathione) 70-18-8; (glutathione disulfide) 27025-41-8; (mannitol) 69-65-8, 87-78-5; (mesna) 19767-45-4, 3375-50-6; (penicillamine) 2219-30-9, 52-67-5; (superoxide dismutase) 37294-21-6, 9016-01-7, 9054-89-1; (thiol derivative) 13940-21-1; (thioneine) 497-30-3; (tiopronin) 1953-02-2
COMPANY NAME: Sigma (Germany); Asta (Germany)

L97 ANSWER 87 OF 90 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 91095948 EMBASE
DOCUMENT NUMBER: 1991095948
TITLE: Chemoprotectants for cancer chemotherapy.
AUTHOR: Dorr R.T.
CORPORATE SOURCE: Arizona Cancer Center, 1515 N Campbell Ave,Tucson, AZ 85724, United States
SOURCE: Seminars in Oncology, (1991) Vol. 18, No. 1 SUPPL. 2, pp. 48-58. .
ISSN: 0093-7754 CODEN: SOLGAV
COUNTRY: United States
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 016 Cancer
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English

SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 16 Dec 1991
Last Updated on STN: 16 Dec 1991

ABSTRACT: Maximal dosing of cytotoxic chemotherapy drugs is often limited by the development of severe nonmyelosuppressive toxicities. Numerous studies have demonstrated that sulfur-containing nucleophiles can antagonize the dose-limiting effects of alkylating agents on the genitourinary tract. Examples include the use of sodium thiosulfate to prevent cisplatin-induced renal tubular necrosis and the use of sulfhydryl-containing compounds like N-acetylcysteine and 2-mercaptoethanesulfonate (mesna) to block oxazophosphorine-induced bladder toxicity. Mesna does not block the antitumor action of oxazophosphorines due to its rapid formation of the inactive dimer ***dimesna*** in the bloodstream. The active monomer is selectively reduced from **dimesna** in renal tubule cells, thereby limiting the inactivation of toxins like acrolein to the genitourinary tract. Recent clinical trials suggest that oral mesna has adequate bioavailability (roughly 50% by urinary thiol measurements) to prevent urotoxicity in high-dose ifosfamide regimens. In addition, mesna is stable in aqueous oral formulations. This may facilitate more convenient oral mesna dosing in protocols using high-dose cyclophosphamide or ifosfamide. Whereas agents like mesna and sodium thiosulfate complex directly with activated (electrophilic) alkylator species, chemoprotectants for the anthracyclines appear to complex with metal cofactors like iron, which are required for the production of cardiotoxicity. Several ethylenediaminetetraacetic-like agents have been evaluated, and a water-soluble piperazinyl derivative, ICRF-187, is currently undergoing clinical evaluation in patients receiving large cumulative doxorubicin doses. An initial clinical trial suggests that ICRF-187 can prevent doxorubicin-induced cardiomyopathy. As with mesna, ICRF-187 does not block the myelosuppressive or the antitumor effects of doxorubicin. Overall, these studies show that site-selective chemoprotection is now feasible for at least two major classes of anticancer agents.

CONTROLLED TERM: Medical Descriptors:
*cancer: DT, drug therapy
*cardiomyopathy: PC, prevention
*cardiomyopathy: SI, side effect
*drug toxicity
conference paper
human
intraperitoneal drug administration
intravenous drug administration
nephrotoxicity: PC, prevention
nephrotoxicity: SI, side effect
oral drug administration
priority journal
Drug Descriptors:
*acetylcysteine: PD, pharmacology
*acetylcysteine: CB, drug combination
*cisplatin: AE, adverse drug reaction
*cisplatin: CB, drug combination
*ifosfamide: DO, drug dose
*ifosfamide: AE, adverse drug reaction
*ifosfamide: CB, drug combination
*mesna: CR, drug concentration
*mesna: PD, pharmacology
*mesna: PK, pharmacokinetics
*mesna: CB, drug combination
*oxazaphosphorine derivative: AE, adverse drug reaction
*oxazaphosphorine derivative: CB, drug combination
*sodium thiosulfate: CB, drug combination

*sodium thiosulfate: PD, pharmacology
allopurinol: CB, drug combination
allopurinol: PD, pharmacology
amifostine: CB, drug combination
amifostine: PD, pharmacology
asparaginase: PD, pharmacology
asparaginase: CB, drug combination
cyclophosphamide
diethyldithiocarbamic acid
dimesna: PD, pharmacology
dimesna: CB, drug combination
doxorubicin: AE, adverse drug reaction
doxorubicin: CB, drug combination
fluorouracil: CB, drug combination
fluorouracil: AE, adverse drug reaction
folinic acid: PD, pharmacology
folinic acid: CB, drug combination
methotrexate: TO, drug toxicity
methotrexate: CB, drug combination
razoxane: PD, pharmacology
razoxane: CB, drug combination
thiourea: CB, drug combination
thiourea: PD, pharmacology
thymidine: CB, drug combination
thymidine: PD, pharmacology
uridine: CB, drug combination
uridine: PD, pharmacology
unclassified drug

CAS REGISTRY NO.: (acetylcysteine) 616-91-1; (cisplatin) 15663-27-1, 26035-31-4, 96081-74-2; (ifosfamide) 3778-73-2; (mesna) 19767-45-4, 3375-50-6; (sodium thiosulfate) 10102-17-7, 7772-98-7, 8052-33-3; (allopurinol) 315-30-0; (amifostine) 20537-88-6; (asparaginase) 9015-68-3; (cyclophosphamide) 50-18-0; (diethyldithiocarbamic acid) 147-84-2, 148-18-5, 3699-30-7, 392-74-5; (**dimesna**) **16208-51-8**, **45127-11-5**; (doxorubicin) 23214-92-8, 25316-40-9; (fluorouracil) 51-21-8; (folinic acid) 58-05-9, 68538-85-2; (methotrexate) 15475-56-6, 59-05-2, 7413-34-5; (razoxane) 21416-67-1, 21416-87-5, 24584-09-6, 24613-06-7; (thiourea) 62-56-6; (thymidine) 50-89-5; (uridine) 58-96-8
CHEMICAL NAME: (1) Cytoxan; Icrf 187; Wr 2721
COMPANY NAME: (1) Bristol; Mead johnson

L97 ANSWER 88 OF 90 EMBASE
ACCESSION NUMBER: 88000585 EMBASE
DOCUMENT NUMBER: 1988000585
TITLE: Mesna - a short review.
AUTHOR: Shaw I.C.; Graham M.I.
CORPORATE SOURCE: Toxicology Laboratory, University College London, London WC1, United Kingdom
SOURCE: Cancer Treatment Reviews, (1987) Vol. 14, No. 2, pp. 67-86.
.
ISSN: 0305-7372 CODEN: CTREDJ
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal
FILE SEGMENT: 016 Cancer
030 Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles

LANGUAGE: English
ENTRY DATE: Entered STN: 11 Dec 1991
Last Updated on STN: 11 Dec 1991
CONTROLLED TERM: Medical Descriptors:
adverse drug reaction
hemorrhagic cystitis
review
human experiment
human
nonhuman
Drug Descriptors:
***protective agent**
mucolytic agent
***dimesna: PD, pharmacology**
***dimesna: PK, pharmacokinetics**
***dimesna: CT, clinical trial**
*mesna: PD, pharmacology
*mesna: PK, pharmacokinetics
*mesna: CT, clinical trial
unclassified drug
CAS REGISTRY NO.: **(dimesna) 16208-51-8, 45127-11-5**; (mesna) 19767-45-4, 3375-50-6
CHEMICAL NAME: (1) Mistabron
COMPANY NAME: (1) Ucb (France)

L97 ANSWER 89 OF 90 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 84170177 EMBASE
DOCUMENT NUMBER: 1984170177
TITLE: Pharmacokinetics and mechanism of action of detoxifying low-molecular-weight thiols.
AUTHOR: Brock N.; Hilgard P.; Pohl J.; et al.
CORPORATE SOURCE: Department of Experimental Cancer Research, Asta-Werke AG, Degussa Pharma Gruppe, D-4800 Bielefeld 14, Germany
SOURCE: Journal of Cancer Research and Clinical Oncology, (1984) Vol. 108, No. 1, pp. 87-97. .
CODEN: JCROD7
COUNTRY: Germany
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
016 Cancer
052 Toxicology
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Dec 1991
Last Updated on STN: 10 Dec 1991
ABSTRACT: A number of thiol compounds have been studied with reference to their selective protective action against urotoxic side-effects of oxazaphosphorine cytostatics. The uroprotective capacity is determined exclusively by the pharmacokinetic behavior of the compound. When given PO, all compounds tested were absorbable from the gut. Both thiols and disulfides are rapidly eliminated from the blood, but during their short half-life a number of unknown chemical reactions probably take place to maintain a physiological redox equilibrium. Elimination from the blood plasma occurs via two fundamentally different mechanisms: by distribution throughout the tissues and intracellular uptake or, alternatively, by rapid renal excretion. Most of the compounds tested belong to the first group: N-acetylcysteine, carboxycysteine, disulfiram and its metabolite DDTC, glutathione, WR 2721, etc. Few compounds are quantitatively excreted through the urine: mesna, ***dimesna*** , and DA 12. Only these compounds were suitable for selective regional detoxification and for the prevention of oxazaphosphorine-induced

urotoxic lesions.

CONTROLLED TERM: Medical Descriptors:
*3 mercapto 2 methylpropionylglycine
*cancer chemotherapy
***dimesna**
*drug absorption
*drug blood level
*drug clearance
*drug comparison
*drug detoxification
*drug efficacy
*drug elimination
*drug half life
*drug interaction
*drug toxicity
*drug urine level
*pharmacokinetics
*urotoxicity
detoxification
intoxication
urinary tract
therapy
intravenous drug administration
oral drug administration
human
normal human
heart
rat
respiratory system
controlled study
prevention
small intestine
liver
kidney
human experiment
animal experiment
animal cell
Drug Descriptors:
*acetylcysteine
***amifostine**
*carbocisteine
*diethyldithiocarbamic acid
*disulfiram
*glutathione
*mesna
*oxazaphosphorine
*sodium thiosulfate
*thiol derivative
oxazaphosphorine derivative
3 mercapto 2 methylpropionylglycine
unclassified drug

CAS REGISTRY NO.: (acetylcysteine) 616-91-1; (amifostine) 20537-88-6; (carbocisteine) 638-23-3; (diethyldithiocarbamic acid) 147-84-2, 148-18-5, 3699-30-7, 392-74-5; (disulfiram) 97-77-8; (glutathione) 70-18-8; (mesna) 19767-45-4, 3375-50-6; (sodium thiosulfate) 10102-17-7, 7772-98-7, 8052-33-3; (thiol derivative) 13940-21-1

CHEMICAL NAME: Da 12; Wr 2721

COMPANY NAME: Asta (Germany); Homburg (Germany); Degussa (Germany); Merck

(Germany); Inpharzam (Germany)

L97 ANSWER 90 OF 90 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:549734 BIOSIS
DOCUMENT NUMBER: PREV200100549734
TITLE: Method of treating inflammatory bowel disorders.
AUTHOR(S): Hausheer, Frederick H. [Inventor, Reprint author]; Peddaiahgari, Seetharamulu [Inventor]
CORPORATE SOURCE: 203 Kendall Pkwy., Boerne, TX, 78229, USA
PATENT INFORMATION: US 6291441 20010918
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Sep. 18, 2001) Vol. 1250, No. 3. e-file. CODEN: OGUPE7. ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
ENTRY DATE: Entered STN: 21 Nov 2001
Last Updated on STN: 25 Feb 2002
ABSTRACT:This invention relates to a method of treating patients suffering from the inflammatory bowel disorders. The method includes administering to a patient in need of treatment an effective amount of a thiol or reducible disulfide compound according to the formula set forth in the specification.
NAT. PATENT. CLASSIF.:514109000
CONCEPT CODE: General biology - Miscellaneous 00532
INDEX TERMS: Major Concepts
Gastroenterology (Human Medicine, Medical Sciences); Methods and Techniques; Pharmacology
INDEX TERMS: Diseases
Crohn's Disease: digestive system disease, immune system disease
Crohn Disease (MeSH)
INDEX TERMS: Diseases
diverticulitis: digestive system disease
Diverticulitis (MeSH)
INDEX TERMS: Diseases
enteritis: digestive system disease, **radiation**-induced
Enteritis (MeSH)
INDEX TERMS: Diseases
enterocolitis: digestive system disease
Enterocolitis (MeSH)
INDEX TERMS: Diseases
inflammatory bowel disorders: digestive system disease
INDEX TERMS: Diseases
ulcerative colitis: digestive system disease
Colitis, Ulcerative (MeSH)
INDEX TERMS: Diseases
vasculitis: vascular disease, intestinal tract
Vasculitis (MeSH)
INDEX TERMS: Chemicals & Biochemicals
dimesna: gastrointestinal-drug; mesna: gastrointestinal-drug
REGISTRY NUMBER: **16208-51-8 (dimesna)**
19767-45-4 (mesna)

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